MAGNESIUM SALTS VOL I #62

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MAGNESIUM SALTS

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Table of Contents

Summary

CHEMICAL INFORMATION

Table of Contents (Cont'd)

Table of Contents (Cont'd)

SUMMARY

Description and Specifications

1. Magnesium carbonate: Magnesium carbonate is a light, white, friable mass or a bulky, white powder. It occurs naturally as the minerals Landsfordite and Magnesite. The formula for magnesium carbonate is approximately $(MgCO_3)_4 \cdot Mg(OH)_2 \cdot 5H_2O$. It is an odorless substance which is practically insoluble in water (1 part in 3300 parts of CO_2-free water), is insoluble in alcohol, and is dissolved by dilute acids with effervescence. An assay of food grade magnesium carbonate should yield the equivalent of not less than 40.0% and not more than 43.5% of MgO.

2. Magnesium chloride: Magnesium chloride, anhydrous and hexahydrate, occurs as colorless, odorless, deliquescent flakes or crystals which are very soluble in water and freely soluble in alcohol. The molecular formulae are $MgCl_2$ (MW=95.23) and $MgCl_2 \cdot 6H_2O$ (MW = 203.30) for the anhydrous and hexahydrate forms, respectively. The anhydrous form melts at 712°, with rapid heating. Food grade magnesium chloride should be not less than 99% and not more than the equivalent of 105% of $MgCl_2 \cdot 6H_2O$.

3. Magnesium hydroxide: Magnesium hydroxide with the molecular formula $Mg(OH)_2$ and molecular weight 58.34, is a white, bulky powder which occurs naturally as the

mineral Brucite. It is soluble in dilute acids and is practically insoluble in water. An assay of food grade magnesium hydroxide should yield not less than 95% $Mg(OH)_2$ after drying. Loss on ignition should be between 30% and 33%.

4. Magnesium oxide: Magnesium oxide, with the molecular formula MgO and molecular weight 40.32, occurs as a very bulky, white powder (light), 5 grams of which occupy 40-50 ml, or as a relatively dense, white powder (heavy), 5 grams of which occupy 10-20 ml. Periclase is a naturally occurring mineral of MgO. The melting point of magnesium oxide is 2800°. It is practically insoluble in water and is insoluble in alcohols. An assay of food grade magnesium oxide should yield not less than 96% MgO after ignition, with not less than 1.5% CaO. Loss on ignition should be not more than 10%.

5. Magnesium phosphate, dibasic: Dibasic magnesium phosphate is a white odorless crystalline powder with the molecular formula $MgHPO_4 \cdot 3H_2O$ and molecular weight 174.36. It occurs naturally as the minerals Newbergite and Phosphor-roesslerite. It has a density of 2.13 and is slightly soluble in water, soluble in dilute acids and is insoluble in alcohol. An assay of food grade dibasic magnesium phosphate should yield not less than 96% of $Mg_2P_2O_7$ calculated on the ignited basis. Loss on ignition should be between 29% and 36%.

6. Magnesium phosphate, tribasic: Tribasic magnesium phosphate, which may contain 4, 5 or 8 molecules of water of hydration, is a white, odorless, tasteless crystalline powder. The pentahydrate occurs most frequently, and the molecular formula for the anhydrous form is $Mg_3(PO_4)_2$ with molecular weight, 262.86. It is insoluble in water but readily soluble in dilute mineral acids. An assay of food grade tribasic magnesium phosphate should yield not less than 98% and not more than the equivalent of 101.5% of $Mg_3(PO_4)_2$, calculated on the ignited basis. Loss on heating should be between 15% and 23% for the tetrahydrate; 20% and 27% for the pentahydrate; and 30% and 37% for the octahydrate.

7. Magnesium stearate: Magnesium stearate with molecular formula $Mg(C_{18}H_{35}O_2)_2$ contains 96% stearic acid with some palmitic acid in the article of commerce. It occurs as a fine, white, bulky powder, having a faint characteristic odor. It is insoluble in water, alcohol and ether and is decomposed by dilute acids. An assay of food grade magnesium stearate should yield not less than 6.8% and not more than 8% of the equivalent of 8% of MgO.

8. Magnesium sulfate: Magnesium sulfate, $MgSO_4 \cdot 7H_2O$, has a molecular weight of 246.47. It occurs as small, colorless crystals, usually needle-like, with a cooling, saline, bitter taste. It occurs naturally as the mineral

Kieserite. It is freely soluble in water giving neutral solutions, is slowly soluble in glycerin, and is sparingly soluble in alcohol. An assay of food grade magnesium sulfate should yield not less than 99.5% $MgSO_4$ after ignition.

Acute Toxicity

Dowden and Bennet (198) found the median tolerance limit in mg/liter (TL_m) for water fleas (Daphnia magna) to be: 3,391 at 25 hours; 3,699 at 50 hours; and 3,484 at 100 hours. All in standard reference water support medium.

The same authors found the TI_m for 3 species of aquatic animals to be: (a) water fleas (Daphnia magna); 963 at 24 hours, 929 at 48 hours, 861 at 72 hours, and 788 at 96 hours, all in glass wool filtered University Lake water support medium. In standard reference water support medium the TI_m was 3,803 at 96 hours. (b) Fish (Lepomis macrochirus); 19,000 at 24 hours; and (c) snail eggs (Lymnaea sp.); 10,530 at 24 hours, 6,525 at 48 hours, 6,300 at 72 hours and 6,250 at 96 hours in glass wool filtered University Lake water support medium.

Barbour and Winter (037) found the LD_{50} for the subcutaneous injection of magnesium chloride in white mice to be 1050 mg/kg BW.

Selisko and Ackerman (681) determined that the LD_{100} for intraperitoneal administration to Wistar albino rats was 100 mg Mg.

The same authors (680) found that the LD_{50}'s for intraperitoneal administration to Wistar albino rats at various concentrations of magnesium chloride were: 100 mg Mg at 6.5 mg/ml; 90 mg Mg at 13.0 mg/ml; and 66 mg Mg at 17.8 mg/ml.

Selisko and Ackerman (682) found that the LD_{50}'s for intraperitoneal administration to Wistar albino rats at various concentrations of magnesium sulfate were: 150 mg Mg at 6.5 mg/ml; 130 mg Mg at 13.0 mg/ml; and 140 mg Mg at 17.8 mg/ml.

Barbour and Taylor (039) found the LD_{50} for subcutaneous administration of magnesium chloride to rabbits to be 725 mg/kg BW.

Short-Term Toxicity

Moinuddin and Lee (515) fed magnesium sulfate in amounts from 0.88 mmole/kg feed to 138 mmole/kg feed to weanling rats for 4 weeks. They observed chronic diarrhea and depressed intake of food. At the highest amount (138 mmole/kg feed), there was a large increase in cecal weight and length. The authors pointed out that this change had also been reported when $MgCl_2$ or $MgCO_3$ were fed in large quantity to rats.

Many magnesium sulfate poisonings were reported in the literature over the past 40 years. Some of the significant aspects of nine such cases are: (1) Thatcher (760) reported the case of a 26-year-old male who died

an hour after accidentally ingesting a large amount of magnesium sulfate solution. The quantity and concentration were not given. The author cautioned that considering other reports of death and toxicity from magnesium sulfate administration, there are individuals for whom even an average dose may be toxic. (2) As reported by Roller (636), an injection of 2 ml of 20% magnesium sulfate caused tetany with respiratory distress in a 21-year-old woman. (3) Byron (113) discussed the cases of 5 young children who died after a strong magnesium sulfate purge was administered. The authors noted that particularly with strong solutions, greater magnesium absorption than ordinarily occurs can take place. (4) Collins and Russell (146) reported a fatality in a 4-year-old resulting from magnesium sulfate absorption. They pointed out that the gastrointestinal membrane is quite permeable to magnesium salts. (5) Stevens and Wolff (735) reported two cases of toxicity, one fatal, resulting from rectal administration of magnesium sulfate. The authors observed that normal kidneys may not remove magnesium sulfate from the blood stream fast enough to prevent amounts toxic to the central nervous system from accumulating. (6) Rosler (641) warned against the peroral administration of large doses of magnesium sulfate (epsom salts) after the deaths of two persons, undergoing a tapeworm cure. He pointed out

that under certain physiological conditions magnesium sulfate is absorbed from the intestinal tract which inundates the system with a large amount of magnesium leading to deep narcosis and paralysis of the respiratory center. (7) Colomb et al. (147) discussed the case of lesions on the cheeks and lips resulting from epsom salts spilled on the face. (8) Rees (620) reported the death of a pregnant young woman who dies from the effects of epsom salts used to induce absortion, which had entered the blood stream via the uterus. (9) Willner (827) described the death of a hospitalized woman accidentally administered magnesium sulfate by duodenal tube.

Allison (012) discussed a toxic sulfate effect from water high in $MgSO_4$ and other sulfates, on both man and livestock. He urged further experimental research particularly concerning the effect on calcium equilibrium.

Hirschfelder (323) studied the effects of oral administration of magnesium sulfate on normal persons and those with renal disease. He concluded that an ordinary epsom salts purgation of 20-30 grams in nephritic patients can induce a magnesium coma easily mistaken for uremic coma. He found that the maximum increase of plasma magnesium was 0.4 mg/100 cc for normal individuals whereas it was 7-9 mg/100 cc in nephritic patients.

Abramowitz and Russo (001) described the case of a patient who developed a rash on her hands after ingesting magnesium hydroxide either as tablets or in suspension.

Brady and Williams (098), Lipsitz and English (448) and Lipsitz (447) all reported magnesium toxicity in newborns whose mothers were administered magnesium sulfate prior to delivery. Stone and Pritchard (736) on the other hand, reported that they had not found any deleterious effects from this treatment.

Long-Term Toxicity

No chronic or long-term toxicity studies could be found in the literature.

Special Studies

Labkovsky (424) found that magnesium salts (chloride or sulfate) administered orally or subcutaneously stimulated the induction of pulmonary adenomas by urethan.

Bazikyan and Akimov (051) studied the antineoplastic effect of magnesium chloride. They found that under the conditions of their experiments and in the amount administered, $MgCl_2$: (1) reduced the toxicity of 9,10-dimethyl-1,2-benzanthracene (DMBA) and 1,2,5,6-dibenzanthracene (DBA); and (2) markedly decreased the number of skin papillomas induced by DMBA and subcutaneous sarcomas induced by DBA.

Wies (826) found that subcutaneous doses of magnesium sulfate solution lowered the fertility of the females and produced defects in the brain and spinal cord of some of the fetuses.

Neal and Neal (537) studied the effect of hard water and water containing magnesium sulfate on atherosclerosis.

They observed that the rabbits given magnesium sulfate were the only group which had no atherosclerosis, did not have diarrhea and were larger with more subcutaneous fat. In addition even though the blood serum of all the experimental groups contained the same amount of fat, only the serums from the magnesium sulfate group were clear, the others were turbid. The authors questioned whether there is a protective substance in hard water or whether there is a specific effect of magnesium on fat metabolism. Further research with other ionizing compounds needs to be done.

Ozsoylu (565) examined the effect of giving magnesium sulfate to control blood sugar levels in 3 diabetic children. It was found that the blood sugar levels of the diabetics dropped (116 mg%, 195 mg% and 234 mg%) within one hour following the infusion of magnesium sulfate. There was no effect on the blood sugar of normal controls.

Frewin et al. (237) found that when intraarterial infusions of magnesium sulfate were given into the brachial artery, there was a direct vasodilator action on the blood vessels of the forearm, as measured by forearm or hand blood flow.

Absorption and Distribution

Barbour and Winter (038) investigated both the absorbability of soluble organic and inorganic magnesium salts

and the subject of magnesium and calcium storage. They performed a number of feeding experiments on dogs from which they concluded: (1) the absorptive capacity of magnesium cannot be regarded as a constant; (2) the form in which magnesium is ingested can determine both its rate and total amount of absorption; (3) both soluble organic and inorganic magnesium salts markedly raise the level of serum magnesium; (4) the retention of calcium, when there is prolonged administration of large amounts of soluble organic magnesium salts, is dependent on the phosphate intake. Adequate phosphate intake leads to no loss of serum calcium while a phosphate intake exceeding 4 mmoles/kg/day leads to retention of additional calcium.

In a review of magnesium metabolism Wacker and Parisi (809) discussed current knowledge of magnesium absorption. The dietary content of anions, particularly phosphate influences the amount of magnesium absorbed. Thus stool content varies both with intake and the anions in the diet. Calcium, by competition for a common absorptive pathway, also effects the amount of absorbed magnesium (Alcock and MacIntyre,(007).

From animal studies it has been found that magnesium is mainly absorbed in the small intestine. Studies in man with ^{28}Mg have shown that peak levels of the isotope are measured in the plasma between two and eight hours

after administration of the dose (Graham et al., 271). The time course of appearance of orally administered labeled magnesium (^{28}Mg) in human plasma is consistent with the finding in animals that the small intestine is the main site of magnesium absorption. However, the development of hypermagnesemia after rectal enemas is evidence for colonic absorption as well (Stevens and Wolff, 735).

Normally about one-third of the ingested magnesium is absorbed and excreted in the urine. Under ordinary circumstances of dietary intake this figure is a good estimation. However, the total dietary intake of this element must be taken into consideration.

In a study (Graham et al., 271) of 13 subjects given oral ^{28}Mg, 44.3% of a diet containing 20 mEq/day was found to be absorbed, whereas 75.8% of a diet containing 1.9 mEq and 23.7% of a diet containing 47 mEq/day were found to be absorbed.

The contention that little endogenous magnesium is eliminated by the fecal route under ordinary circumstances is supported by the observation that when ^{28}Mg is given intravenously to normal subjects, only 1-2% is recovered in the feces (Aikawa et al., 005 and Silver et al., 701).

Metabolism and Excretion

Clark (142) studied the effects of orally administered magnesium chloride on calcium and phosphate metabolism in adult rats. The authors concluded that: (1) dietary magnesium ions markedly influenced the absorption and excretion of calcium and phosphorus; (2) renal conservation of phosphorus is responsible for the improvement in phosphorus balance resulting from supplemental dietary magnesium; (3) two possible consequences for human beings might be: (a) in the treatment or prevention of those metabolic bone diseases resulting from inadequate intestinal absorption of calcium and (b) the observation that supplementary dietary magnesium which resulted in a drop in urinary phosphorus could serve as a partial explanation for the reduction of renal calculus formation following oral administration of magnesium salts.

Massry et al. (486) evaluated the characteristics of the renal handling of magnesium in dogs. They found that magnesium excretion is determined by filtration and reabsorption alone without evidence for tubular secretion.

Smith et al. (712) studied the excretion of magnesium ions in dogs following the intravenous injection of magnesium sulfate. They concluded that during the first 3-4 hours the magnesium ions were distributed throughout the extracellular fluid, while subsequently indications were that some of the ions were segregated from the extracellular fluid and thus not excreted.

Aikawa et al. (005) used ^{28}Mg to investigate the kinetics of magnesium distribution in normal and diseased subjects. Following intravenous administration the isotope rapidly appeared in the urine but only insignificant amounts appeared in the stool. This was taken to indicate that most of the magnesium in the stool is of exogenous origin. Equilibration of the isotope with magnesium in the body was slow and parenterally administered magnesium was excreted very slowly by the kidneys. There was no marked difference between the results obtained from healthy and diseased patients.

Consolazio et al. (151) studied magnesium excretion in sweat. They concluded that under extreme conditions sweat could account for about 25% of the daily magnesium loss.

Figure 1 in Biochemical Section III, D 4 shows those enzyme systems in intermediary metabolism known to require magnesium (Ozsoylu, 565).

Drake (200) attempted to ascertain whether magnesium oxide exerts a "protective effect" in calcium oxolate stone formation. The test group (daily oral administration of MgO) was found to excrete significantly more calcium than the controls.

Enzymes and Other Biochemical Parameters

Both MacIntyre (463) and Wacker and Parisi (809) summarized the in vitro actions of magnesium ions (see main body of text for details). MacIntyre noted that

the reactions involving ATP and ADP which are activated by magnesium are so widespread that magnesium must influence all life processes. Wacker and Parisi (809) concluded that since magnesium is the most abundant divalent intracellular cation, that it would be a tenable hypothesis to assume analogous in vivo function.

LaBarre and Vesselovsky (421) investigated the influence of magnesium sulfate on the functioning of the exocrine portion of the pancreas. They concluded that: (1) magnesium sulfate has a depressive effect on gastric and pancreatic secretions; (2) this effect is dependent on the integrity of the encephalic centers. Thus in those persons whose pancreatic function is curtailed either by secretin administration or the presence of food in the digestive tract, magnesium sulfate causes a reduction in production of pancreatic juice and enzyme formation.

Drug Interaction

Barbour and Taylor (039) studied the relations obtained various combinations of sodium barbital and magnesium chloride with respect to toxicity and hypnotic action. They concluded that the combination of magnesium chloride and sodium barbital hastened the onset of narcosis and lessened the persistence of the narcotic effect without significantly increasing toxicity.

Barbour and Winter (037) and Winter and Barbour (831) investigated the effect of magnesium chloride on the antipyretic action of amidopyrine and sodium salicylate.

Their observations with respect to both these experiments were: (1) various combinations of magnesium chloride with amidopyrine showed both higher antipyretic action and lower toxicity than amidopyrine alone; (2) the combination of 150 mg/kg BW magnesium chloride with 50 mg/kg BW sodium salicylate was more effective than 100 mg/kg BW sodium salicylate alone. The authors concluded that magnesium salts potentiate the early effects of antipyretic drugs even when intense or prolonged action tends to be inhibited.

Winter et al. (832) studied the effect of magnesium oxide on the antipyretic action of phenacetin. They found that the antipyretic action of orally administered phenacetin was enhanced by the addition of MgO. Since MgO alone was not antipyretic they considered their results to be strongly indicative of a true synergism.

Gershoff and Prien (253) found that after 5 years, 30 out of 36 patients with recurring calcium oxalate urolithiasis, maintained on a daily oral program of magnesium oxide and vitamin B_6, showed either no recurrence or decreased recurrence of stone formation. The authors postulated that a possible explanation for the observed prevention of renal stone formation might involve an effect on the solvent characteristics of urine.

Consumer Exposure

Magnesium is a natural constituent of all vegetables, fruits, meats and seafoods. (See Tables in main body of text.) The magnesium compound most widely used for both food processing and pharmaceutical purposes, is magnesium carbonate (NAS/NRC Tables). In food processing it serves as: an anti-caking and drying agent, a carrier, a disintegrating and dispersing agent, a color fixative, a color retention agent and as an adjunct. Pharmaceutically, magnesium carbonate is used as an antacid and a laxative. Additional uses for other magnesium salts are: magnesium chloride (831) as an emulsifying agent for suspending dried skim milk in water; magnesium oxide (610) for clarifying sugar juice and in molasses and raw sugar processing; magnesium phosphates as nutrients and dietary supplements; and magnesium stearate (405) as a foaming and whipping agent and in plasticizers used for food packaging materials.

Hard water is another source of exposure to magnesium.

Magnesium has been recognized as an essential nutrient since 1932. However, as a major inorganic constituent of living things and as a major intracellular cation, it is not in the strictest sense a nutrient (677).

According to Schroeder et al. (677): (1) except in states of malabsorption and excessive excretion due to other conditions, there is no evidence of widespread dietary magnesium deficiency; (2) the daily dietary

requirement for adult man is probably the obligatory urinary excretion plus losses in sweat and the amount unabsorbed in feces; (3) there is no good evidence that marginal dietary intakes contribute to any chronic disease of unknown etiology.

CHEMICAL INFORMATION

MAGNESIUM CARBONATE

I. Nomenclature

A. Common names: Landsfordite, Magnesite (Magnesium carbonate minerals)

B. Chemical names: Magnesium carbonate, Magnesium carbonate hydroxide

C. Trade names: none

D. Chemical Abstracts Services Unique Registry Number: 1319502

II. Empirical Formula

Approximately $O_{14}Mg_5C_4H_2 \cdot 5OH_2$

III. Structural Formula

Approximately $(MgCO_3)4 \cdot Mg(OH)_2 \cdot 5H_2O$

IV. Molecular Weight: 485.69 (based on approximated formula)

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade magnesium carbonate:

1. Description

Magnesium carbonate is a basic hydrated magnesium carbonate or a normal hydrated magnesium carbonate. It occurs as light, white, friable masses, or as a bulky, white powder. It is odorless, and is stable in air. It is practically insoluble in water, to which, however, it imparts a slightly alkaline reaction. It is insoluble in alcohol, but is dissolved by dilute acids with effervescence. When treated with diluted

hydrochloric acid T.S., it dissolves with effervescence and the resulting solution gives positive tests for Magnesium.

2. Specifications

Assay. The equivalent of not less than 40% and not more than 43.5% of MgO.

Limits of Impurities

Acid-insoluble substances. Not more than 500 parts per million (0.5%).

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Calcium oxide. Not more than 0.6%.

Heavy metals (as Pb). Not more than 30 parts per million (0.003%).

Lead. Not more than 10 parts per million (0.001%).

Soluble salts. Not more than 1%.

3. Tests

Assay. Dissolve about 1 gram, accurately weighed, in 30 ml of 1 N sulfuric acid, add methyl orange T.S., and titrate the excess acid with 1 N sodium hydroxide. From the volume of 1 N sulfuric acid consumed, deduct the volume of 1 N sulfuric acid corresponding to the content of calcium oxide in the weight of the sample taken for the assay. The difference is the volume of 1 N sulfuric acid equivalent to the magnesium oxide present. Each ml of 1 N sulfuric acid is equivalent to 20.16 mg of MgO and to 28.04 mg of CaO.

Acid-insoluble substances. Mix 5 grams with 75 ml of water, add hydrochloric acid in small portions, with agitation, until no more of the sample dissolves, and boil for 5 minutes.

If an insoluble residue remains, filter, wash well with water until the last washing is free from chloride, ignite, cool, and weigh.

Arsenic. A solution of 1 gram in 10 ml of diluted hydrochloric acid T.S. meets the requirements of the Arsenic Test.

Calcium oxide. Dissolve about 1 gram, accurately weighed, in a mixture of 3 ml of sulfuric acid and 22 ml of water. Add 50 ml of alcohol, and allow the mixture to stand overnight. If crystals of magnesium sulfate separate, warm the mixture to about 50° to dissolve them. Filter through a Gooch crucible containing an asbestos mat that previously has been washed with diluted sulfuric acid T.S., water, and alcohol, and ignited and weighed. Wash the crystals on the mat several times with a mixture of 2 volumes of alcohol and 1 volume of diluted sulfuric acid T.S. Ignite the crucible and contents at a dull red heat, cool, and weigh. The weight of calcium sulfate so obtained, multiplied by 0.4119, gives the equivalent of calcium oxide in the sample taken for the test.

Heavy metals. Dissolve 667 mg in 10 ml of diluted hydrochloric acid T.S., and evaporate the solution to dryness on a steam bath. Toward the end of the evaporation stir frequently to disintegrate the residue so that finally a dry powder is obtained. Dissolve the residue in 20 ml of water, and evaporate to dryness in the same manner as before. Redissolve the residue in 25 ml of water, and filter if necessary. This solution meets the requirements of the Heavy Metals Test, using 20 mcg of lead ion (Pb) in the control (Solution A).

Lead. A solution of 1 gram in 10 ml of diluted hydrochloric acid T.S. meets the requirements of the Lead Limit Test, using 10 mcg of lead ion (Pb) in the control.

Soluble salts. Mix 2 grams with 100 ml of a mixture of equal volumes of *n*-propyl alcohol and water. Heat the mixture to the boiling point with constant stirring, cool to room temperature, add water to make 100 ml, and filter. Evaporate 50 ml of the filtrate on a steam bath to dryness, and dry at 105° for 1 hour. The weight of the residue does not exceed 10 mg.

Packaging and storage. Store in well-closed containers.

Functional use in foods. Alkali; drying agent; color-retention agent; anticaking agent; carrier.

VI. Description

A. General characteristics

Magnesium carbonate occurs as an odorless, light, white, friable mass, or white, bulky powder.

B. Physical properties

Magnesium carbonate is soluble in 3300 parts of CO_2-free water, to which it imparts an alkaline reaction, and is more soluble in water containing CO_2. It is insoluble in alcohol and soluble in weak acids with effervescence.

C. Stability in containers

Not available.

MAGNESIUM CHLORIDE

I. Nomenclature

A. Common names: none

B. Chemical names: Magnesium chloride, Magnesium chloride (hexahydrate)

C. Trade names: Magnogene

D. Chemical Abstracts Services Unique Registry Number:

7786303 (anhydrous)

7791186 (hexahydrate)

II. Empirical Formula

Cl_2Mg (anhydrous)

III. Structural Formula

$MgCl_2$; $MgCl_2 \cdot 6H_2O$

IV. Molecular Weight: 95.23 (anhydrous), 203.30 (hexahydrate)

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade magnesium chloride:

1. Description

Colorless, odorless flakes or crystals. It is very deliquescent. It is very soluble in water and freely soluble in alcohol. A 1 in 10 solution gives positive tests for Magnesium, and for Chloride.

2. Specifications

Assay. Not less than 99% and not more than the equivalent of 105% of $MgCl_2 \cdot 6H_2O$.

Limits of Impurities

Ammonium. Not more than 50 parts per million (0.005%).

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Heavy metals (as Pb). Not more than 10 parts per million (0.001%).

Sulfate. Not more than 200 parts per million (0.02%).

3. Tests

Assay. Dissolve about 450 mg, accurately weighed, in 25 ml of water, add 5 ml of ammonia-ammonium chloride buffer T.S. and 0.1 ml of eriochrome black T.S., and titrate with 0.05 M disodium ethylenediaminetetraacetate until the solution is blue in color. Each ml of 0.05 M disodium ethylenediaminetetraacetate is equivalent to 10.16 mg of $MgCl_2 \cdot 6H_2O$.

Ammonium. Dissolve 1 gram in 90 ml of water, and slowly add 10 ml of a freshly boiled and cooled solution of sodium hydroxide (1 in 10). Allow to settle, then decant 20 ml of the supernatant liquid into a color comparison tube, dilute to 50 ml with water, and add 2 ml of Nessler's reagent. Any color does not exceed that produced by 10 mcg of ammonium (NH_4) ion in 48 ml of water and 2 ml of the sodium hydroxide solution.

Arsenic. A solution of 1 gram in 35 ml of water meets the requirements of the Arsenic Test.

Heavy metals. A solution of 2 grams in 25 ml of water meets the requirements of the Heavy Metals Test, using 20 mcg of lead ion (Pb) in the control (Solution A).

Sulfate. Any turbidity produced by a 1-gram sample does not exceed that shown in a control containing 200 mcg of sulfate (SO_4).

Packaging and storage. Store in tight containers.

Functional use in foods. Color-retention agent; firming agent.

VI. Description

A. General characteristics

Magnesium chloride and its hexahydrate occur as colorless, odorless flakes or crystals which are deliquescent.

B. Physical properties

1. Anhydrous. The density of the anhydrous form is 2.41 (also reported as 2.325) and the melting point is 712° (rapid heating). It is soluble in water with the evolution of much heat and forming clear solutions.

2. Hexahydrate. The density of the hexahydrate form of magnesium chloride is 1.56. It loses $2 \cdot H_2O$ at 100°C, and begins to lose some HCl at 110°C. One gram is soluble in 0.6 ml of water, 0.3 ml of boiling water, and 2 ml of alcohol.

C. Stability in containers

Not available.

MAGNESIUM HYDROXIDE

I. Nomenclature

A. Common names: Brucite (naturally occuring mineral)

B. Chemical names: Magnesium hydroxide, Magnesium hydrate

C. Trade names: Marinco-H, Hydro-Magma (30% suspension of magnesium hydroxide in water)

D. Chemical Abstracts Services Unique Registry Number: 1309428

II. Empirical Formula

O_2MgH_2

III. Structural Formula

$Mg(OH)_2$

IV. Molecular Weight: 58.34

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade magnesium hydroxide:

1. Description

A white, bulky powder. It dissolves in dilute acids, but is practically insoluble in water and in alcohol. A 1 in 20 solution in diluted hydrochloric acid T.S. gives positive tests for magnesium.

2. Specifications

Assay. Not less than 95% of $Mg(OH)_2$ after drying.

Loss on ignition. Between 30% and 33%.

Limits of Impurities

Alkalies (free) and soluble salts. Passes test.

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Calcium oxide. Not more than 1%.

Heavy metals (as Pb). Not more than 40 parts per million (0.004%).

Lead. Not more than 10 parts per million (0.001%).

Loss on drying. Not more than 2%.

3. Tests

Assay. Transfer about 400 mg, previously dried at 105° for 2 hours and accurately weighed, into an Erlenmeyer flask. Add 25 ml of 1 N sulfuric acid, and, after solution is complete, add methyl red T.S. and titrate the excess acid with 1 N sodium hydroxide. From the volume of 1 N sulfuric acid consumed, deduct the volume of 1 N sulfuric acid corresponding to the content of calcium oxide in the sample taken for the assay. The difference is the volume of 1 N sulfuric acid equivalent to the $Mg(OH)_2$ in the sample of magnesium hydroxide taken. Each ml of 1 N sulfuric acid is equivalent to 29.16 mg of $Mg(OH)_2$ and to 28.04 mg of CaO.

Loss on ignition. Transfer about 500 mg, accurately weighed, to a tared platinum crucible, and ignite, increasing the heat gradually, to constant weight.

Alkalies (free) and soluble salts. Boil 2 grams with 100 ml of water for 5 minutes in a covered beaker, then filter while hot. Titrate 50 ml of the cooled filtrate with 0.1 N sulfuric acid, using methyl red T.S. as the indicator. Not more than 2 ml of the acid is consumed. Evaporate 25 ml of the filtrate to dryness, and dry at 105° for 3 hours. Not more than 10 mg of residue remains.

Arsenic. A solution of 1 gram in 25 ml of diluted hydrochloric acid T.S. meets the requirements of the Arsenic Test.

Calcium oxide. Dissolve about 500 mg, accurately weighed, in a mixture of 3 ml of sulfuric acid and 22 ml of water. Add 50 ml of alcohol, and allow the mixture to stand overnight. Warm the mixture to about 50°, if necessary, to dissolve any crystals of magnesium sulfate, and filter through a Gooch crucible containing an asbestos mat which has been previously washed with diluted sulfuric acid T.S., water, and alcohol, and ignited. Wash the crystals on the mat several times with a mixture of 3 volumes of alcohol and 1 volume of water. Ignite the crucible and contents at a dull red heat, cool, and weigh. The weight of calcium sulfate thus obtained, multiplied by 0.4119, gives the equivalent of calcium oxide (CaO).

Heavy metals. Dissolve 1 gram in 10 ml of diluted hydrochloric acid T.S., and evaporate to dryness on a steam bath. Toward the end of the evaporation, stir the residue frequently, disintegrate it to obtain a dry powder, dissolve the powder in 20 ml of water, and filter. A 10-ml portion of the filtrate meets the requirements of the Heavy Metals Test, using 20 mcg of lead ion (Pb) in the control (Solution A).

Lead. A solution of 1 gram in 20 ml of diluted hydrochloric acid T.S., meets the requirements of the Lead Limit Test, using 10 mcg of lead ion (Pb) in the control.

Loss on drying. Dry at 105° for 2 hours.

Packaging and storage. Store in tight containers.

Functional use in foods. Alkali; drying agent; color-retention agent.

VI. Description

A. General characteristics

Magnesium hydroxide occurs as a white bulky amorphous powder.

B. Physical properties

Magnesium hydroxide is soluble in dilute acids, but is practically insoluble in water (1:80,000). The pH of an aqueous slurry is 9.5-10.5.

C. Stability in containers

Not available.

MAGNESIUM OXIDE

I. Nomenclature

A. Common names: Magnesia, Calcined magnesia, Magnesia usta, Periclase (naturally occuring mineral)

B. Chemical names: Magnesium oxide

C. Trade names: Magcal, Maglite

D. Chemical Abstracts Services Unique Registry Number: 1309484

II. Empirical Formula

OMg

III. Structural Formula

MgO

IV. Molecular Weight: 40.32

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade magnesium oxide:

1. Description

A very bulky, white powder known as light magnesium oxide or a relatively dense, white powder known as heavy magnesium oxide. Five grams of light magnesium oxide occupy a volume of approximately 40-50 ml, while 5 grams of heavy magnesium oxide occupy a volume of approximately 10-20 ml. It is practically insoluble in water and is insoluble in alcohol. It is soluble in dilute acids. A solution of magnesium oxide in diluted hydrochloric acid T.S. gives positive tests for Magnesium.

2. Specifications

Assay. Not less than 96% of MgO after ignition.

Limits of Impurities

Acid-insoluble substances. Not more than 0.1%.

Alkalies (free) and soluble salts. Passes test.

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Calcium oxide. Not more than 1.5%.

Heavy metals (as Pb). Not more than 40 parts per million (0.004%).

Lead. Not more than 10 parts per million (0.001%).

Loss on ignition. Not more than 10%.

3. Tests

Assay. Ignite about 500 mg to constant weight at 800° to 825° in a tared platinum crucible, weigh the residue accurately, dissolve it in 30 ml of 1 N sulfuric acid, boil gently to remove any carbon dioxide, cool, add methyl orange T.S., and titrate the excess acid with 1 N sodium hydroxide. From the volume of 1 N sulfuric acid consumed deduct the volume of 1 N sulfuric acid corresponding to the content of calcium oxide in the magnesium oxide taken for the assay. The difference is the volume of 1 N sulfuric acid equivalent to the MgO in the portion of magnesium oxide taken. Each ml of 1 N sulfuric acid is equivalent to 20.15 mg of MgO and to 28.04 mg of CaO.

Acid-insoluble substances. Mix 2 grams with 75 ml of water, add hydrochloric acid in small portions, with agitation, until no more dissolves, and boil for 5 minutes. If an insoluble residue remains, filter, wash well with water until the last washing is free from chloride, ignite, cool, and weigh.

Alkalies (free) and soluble salts. Boil 2 grams with 100 ml of water for 5 minutes in a covered beaker, and filter while hot. Add methyl red T.S., and titrate 50 ml of the cooled filtrate with 0.1 N sulfuric acid. Not more than 2 ml of the acid is consumed. Evaporate 25 ml of the filtrate to dryness, and dry at 105° for 1 hour. Not more than 10 mg of residue remains.

Arsenic. A solution of 1 gram in 20 ml of diluted hydrochloric acid T.S. meets the requirements of the Arsenic Test.

Calcium oxide. Dissolve about 400 mg, accurately weighed, in a mixture of 3 ml of sulfuric acid and 22 ml of water. Add 50 ml of alcohol, and allow the mixture to stand overnight. If crystals of magnesium sulfate separate, warm the mixture to about 50° to dissolve them. Filter through a Gooch crucible containing an asbestos mat that previously has been washed with diluted sulfuric acid T.S., water, and alcohol, and ignited and weighed. Wash the crystals on the mat several times with a mixture of 2 volumes of alcohol and 1 volume of diluted sulfuric acid T.S. Ignite the crucible and contents at a dull red heat, cool, and weigh. The weight of calcium sulfate obtained, multiplied by 0.4119, gives the equivalent of calcium oxide in the sample taken for the test.

Heavy metals. Dissolve 500 mg in 20 ml of diluted hydrochloric acid T.S., and evaporate the solution to dryness on a steam bath. Toward the end of the evaporation stir frequently to disintegrate the residue so that finally a dry powder is obtained. Dissolve the residue in 20 ml of water and evaporate to dryness in the

same manner as before. Redissolve the residue in 20 ml of water and filter if necessary. This solution meets the requirements of the Heavy Metals Test, using 20 mcg of lead ion (Pb) in the control (Solution A).

Lead. A solution of 1 gram in 20 ml of diluted hydrochloric acid T.S. meets the requirements of the Lead Limit Test, using 10 mcg of lead ion (Pb) in the control.

Loss on ignition. Weigh accurately about 500 mg in a tared covered platinum crucible. Ignite at between 800° and 825° for 15 minutes, cool and weigh.

Packaging and storage. Store in tight containers.

Labeling. Label magnesium oxide to indicate whether it is light magnesium oxide or heavy magnesium oxide.

Functional use in foods. Alkali; neutralizer.

VI. Description

A. General characteristics

Magnesium oxide occurs as a very bulky, white powder (light form) or as a dense, white powder (heavy form). (Five grams of the light form occupies 40-50 ml; 5 grams of the heavy form occupies 10-20 ml.)

B. Physical properties

Magnesium oxide has a melting point of 2800°, is practically insoluble in water and is insoluble in alcohols. It is soluble in dilute acids. The pH of a saturated aqueous solution is 10.3.

C. Stability in containers

Not available.

MAGNESIUM PHOSPHATE, DIBASIC

I. Nomenclature

A. Common names: Newbergite, Phosphor-roesslerite (naturally occuring minerals)

B. Chemical names: Magnesium phosphate, dibasic, Magnesium hydrogen phosphate, Secondary magnesium phosphate

C. Trade names: none

D. Chemical Abstracts Services Unique Registry Number: 7782754

II. Empirical Formula

$HO_4MgP \cdot 3OH_2$

III. Structural Formula

$MgHPO_4 \cdot 3H_2O$

IV. Molecular Weight: 174.36

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade dibasic magnesium phosphate:

1. Description

A white, odorless crystalline powder. It is slightly soluble in water and insoluble in alcohol, but is soluble in dilute acids.

2. Identification

A. Dissolve about 200 mg in 10 ml of diluted nitric acid T.S. and add, dropwise, ammonium molybdate T.S. A greenish yellow precipitate of ammonium phosphomolybdate forms which is soluble in ammonia T.S.

B. Dissolve 100 mg in 0.5 ml of diluted acetic acid T.S. and 20 ml of water. Add 1 ml of ferric chloride T.S., let stand for

5 minutes, and filter. The filtrate gives a positive test for Magnesium.

3. Specifications

Assay. Not less than 96% of $Mg_2P_2O_7$ calculated on the ignited basis.

Loss on ignition. Between 29 and 36%.

Limits of Impurities

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Fluoride. Not more than 10 parts per million (0.001%).

Heavy metals (as Pb). Not more than 30 parts per million (0.003%).

Lead. Not more than 5 parts per million (0.0005%).

4. Tests

Assay. Weigh accurately about 500 mg of the residue obtained in the test for Loss on ignition, dissolve it in a mixture of 50 ml of water and 1 ml of hydrochloric acid, dilute to 100 ml with water, and mix. Transfer 50 ml of this solution into a 250 ml Erlenmeyer flask, add 10 ml of ammonia-ammonium chloride buffer T.S. and 12 drops of eriochrome black T.S., and titrate with 0.1 M disodium ethylenediaminetetraacetate until the wine-red color changes to pure blue. Each ml of 0.1 M disodium ethylenediaminetetraacetate is equivalent to 22.25 mg of $Mg_2P_2O_7$.

Loss on ignition. Weigh accurately about 1 gram, and ignite, preferably in a muffle furnace, at 800° ± 25° to constant weight.

Arsenic. A solution of 1 gram in 5 ml of diluted hydrochloric acid T.S. meets the requirements of the Arsenic Test.

Fluoride. Transfer 5 grams of the sample into a 200-ml distilling flask connected with a condenser and carrying a thermometer and a dropping funnel equipped with a stopcock. Dissolve the sample in 25 ml of dilute sulfuric acid (1 in 4), add 6 glass beads, and connect the apparatus for distillation, using a 600-ml beaker to collect the distillate. Add 40 ml of the dilute sulfuric acid to the flask through the dropping funnel, then fill the funnel with water, heat the solution to boiling, and continue heating until the temperature reaches 165°. Adjust the stopcock of the dropping funnel so that the temperature is maintained at 165° ± 5°, and continue the distillation until about 300 ml has been collected. Rinse the condenser and condenser arm with water, collecting the rinsings in the beaker. Add sodium hydroxide T.S. to the distillate to make it alkaline to litmus paper, and then add 5 ml in excess. Add 5 ml of 30% hydrogen peroxide and 6 glass beads to the beaker, boil until a volume of about 30 ml is reached, and cool. Transfer the condensed distillate, including the glass beads, into a 125-ml distilling flask connected with a condenser and carrying a thermometer and a capillary tube, both of which must extend into the liquid. Add 30 ml of perchloric acid, and continue as directed under the Fluoride Limit Test, Method I, beginning with "Connect a small dropping funnel or a steam generator to the capillary tube."

Heavy metals. Suspend 1.33 grams in 20 ml of water, and add hydrochloric acid, dropwise, until the sample just dissolves. Adjust the pH to between 3 and 4, filter, and dilute the filtrate

to 40 ml with water. For the control (Solution A), add 20 mcg of lead ion (Pb) to 10 ml of the filtrate, and dilute to 40 ml. For the sample (Solution B), dilute the remaining 30 ml of the filtrate to 40 ml. Add 10 ml of hydrogen sulfide T.S. to each solution, and allow to stand for 5 minutes. Solution B is no darker than Solution A.

Lead. Dissolve 1 gram in 20 ml of diluted hydrochloric acid T.S., evaporate the solution to a volume of about 10 ml on a steam bath, dilute to about 20 ml with water, and cool. This solution meets the requirements of the Lead Limit Test, using 5 mcg of lead ion (Pb) in the control.

Packaging and storage. Store in well-closed containers.

Functional use in foods. Nutrient; dietary supplement.

VI. Description

A. General characteristics

Dibasic magnesium phosphate occurs as a white, odorless crystalline powder.

B. Physical properties

Dibasic magnesium phosphate has a density of 2.13; it is slightly soluble in water, soluble in dilute acids, and is insoluble in alcohol.

C. Stability in containers

Not available.

MAGNESIUM PHOSPHATE, TRIBASIC

I. Nomenclature

A. Common names: Bobierrite (naturally occuring mineral)

B. Chemical names: Magnesium phosphate, tribasic, "Neutral" magnesium phosphate, Tertiary magnesium phosphate, Trimagnesium phosphate

C. Trade names: none

D. Chemical Abstracts Services Unique Registry Number: 10233871

II. Empirical Formula

$O_8Mg_3P_4 \cdot 5OH_2$

III. Structural Formula

$Mg_3(PO_4)_2 \cdot 5H_2O$ (also occurs as tetrahydrate and octahydrate)

IV. Molecular Weight: 353.00 (pentahydrate), 262.86 (anhydrous)

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade tribasic magnesium phosphate:

1. Description

Tribasic magnesium phosphate may contain 4, 5, or 8 molecules of water of hydration. It occurs as a white, odorless, tasteless crystalline powder. It is readily soluble in dilute mineral acids but is almost insoluble in water.

2. Identification

A. Dissolve about 200 mg in 10 ml of diluted nitric acid T.S. and add, dropwise, ammonium molybdate T.S. A greenish-yellow precipitate of ammonium phosphomolybdate forms which is soluble in ammonia T.S.

B. Dissolve 100 mg in 0.7 ml of diluted acetic acid T.S. and 20 ml of water. Add 1 ml of ferric chloride T.S., let stand for 5 minutes, and filter. The filtrate gives a positive test for Magnesium.

3. Specifications

Assay. Not less than 98% and not more than the equivalent of 101.5% of $Mg_3(PO_4)_2$, calculated on the ignited basis.

Titration value. Passes test.

Loss on heating. $Mg_3(PO_4)_2 \cdot 4H_2O$, between 15 and 23%; $Mg_3(PO_4)_2 \cdot 5H_2O$, between 20 and 27%; $Mg_3(PO_4)_2 \cdot 8H_2O$, between 30 and 37%.

Limits of Impurities

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Fluoride. Not more than 10 parts per million (0.001%).

Heavy metals (as Pb). Not more than 30 parts per million (0.003%).

Lead. Not more than 5 parts per million (0.0005%).

4. Tests

Assay. Weigh accurately about 200 mg of the sample, and dissolve it in a mixture of 25 ml of water and 10 ml of diluted nitric acid T.S. Filter, if necessary, wash any precipitate, then dissolve the precipitate by the addition of 1 ml of diluted nitric acid T.S. Adjust the temperature to about 50°, add 75 ml of ammonium molybdate T.S., and maintain the temperature at about 50° for 30 minutes, stirring occasionally. Allow to stand for 16 hours or overnight at room temperature. Wash the precipitate once or twice with water by decantation, using from 30-40 ml

each time, and pour these two washings through a filter. Transfer the precipitate to the same filter, and wash with potassium nitrate solution (1 in 100) until the last washing is not acid to litmus paper. Transfer the precipitate and filter to the precipitation vessel, add 50 ml of 1 N sodium hydroxide, agitate until the precipitate is dissolved, add 3 drops of phenolphthalein T.S., and then titrate the excess alkali with 1 N sulfuric acid. Each ml of 1 N sodium hydroxide is equivalent to 5.714 mg of $Mg_3(PO_4)_2$.

Titration value. Ignite about 3 grams at about 425° to constant weight, and dissolve, by warming, 2 grams of the ignited salt in 50 ml of 1 N hydrochloric acid. Cool, add methyl orange T.S., and slowly titrate the excess of 1 N hydrochloric acid with 1 N sodium hydroxide to a yellow color, shaking the mixture vigorously during the titration. Not less than 29 ml and not more than 30.8 ml of 1 N hydrochloric acid is consumed.

Loss on heating. Weigh accurately about 1 gram and heat at about 425° to constant weight.

Arsenic. A solution of 1 gram in 10 ml of diluted hydrochloric acid T.S. meets the requirements of the Arsenic Test.

Fluoride. Determine as directed in the Fluoride Limit Test under Magnesium Phosphate, Dibasic.

Heavy metals. Suspend 1.33 grams in 20 ml of water, and add hydrochloric acid, dropwise, until the sample just dissolves. Adjust the pH to between 3 and 4, filter, and dilute the filtrate to 40 ml with water. For the control (Solution A), add 20 mcg

of lead ion (Pb) to 10 ml of the filtrate, and dilute to 40 ml. For the sample (Solution B), dilute the remaining 30 ml of the filtrate to 40 ml. Add 10 ml of hydrogen sulfide T.S. to each solution, and allow to stand for 5 minutes. Solution B is no darker than Solution A.

Lead. Dissolve 1 gram in 20 ml of diluted hydrochloric acid T.S., evaporate the solution to a volume of about 10 ml on a steam bath, dilute to about 20 ml with water, and cool. This solution meets the requirements of the Lead Limit Test, using 5 mcg of lead ion (Pb) in the control.

Packaging and storage. Store in well-closed containers.

Functional use in foods. Nutrient; dietary supplement.

VI. Description

A. General characteristics

Tribasic magnesium phosphate, containing 4, 5, or 8 molecules of water of hydration, occurs as a white, odorless, tasteless crystalline powder.

B. Physical properties

Tribasic magnesium phosphate is insoluble in water, but readily soluble in dilute mineral acids. It becomes anhydrous at 400°.

C. Stability in containers

Not available.

MAGNESIUM STEARATE

I. Nomenclature

A. Common names: none

B. Chemical names: Magnesium stearate

C. Trade names: none

D. Chemical Abstracts Services Unique Registry Number: 557040

II. Empirical Formula

Approximately $O_4C_{36}H_{70}Mg$

III. Structural Formula

Approximately $Mg(C_{18}H_{35}O_2)_2$, containing 96% stearic acid and some palmitic acid in the article of commerce.

IV. Molecular Weight: Approximately 590

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade magnesium stearate:

1. Description

Magnesium stearate is a compound of magnesium with variable proportions of stearic and palmitic acids. It occurs as a fine, white, bulky, powder, having a faint, characteristic odor. It is unctuous, and is free from grittiness. It is insoluble in water, in alcohol, and in ether. It conforms to the regulations of the federal Food and Drug Administration pertaining to specifications for salts of fatty acids and fatty acids derived from edible fats sources.

2. Identification

A. Heat 1 gram with a mixture of 25 ml of water and 5 ml of hydrochloric acid. Fatty acids are liberated, floating as an oily layer on the surface of the liquid. The water layer gives positive tests for Magnesium.

B. Mix 25 grams of the sample with 200 ml of hot water, then add 60 ml of diluted sulfuric acid T.S., and heat the mixture, with frequent stirring, until the fatty acids separate cleanly as a transparent layer. Wash the fatty acids with boiling water until free from sulfate, collect them in a small beaker, and warm on a steam bath until the water has separated and the fatty acids are clear. Allow the acids to cool, pour off the water layer, then melt the acids, filter into a dry beaker, and dry at 105° for 20 minutes. The solidification point of the fatty acids so obtained is not below 54°.

3. Specifications

Assay. Not less than the equivalent of 6.8% and not more than the equivalent of 8% of MgO.

Limits of Impurities

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Heavy metals (as Pb). Not more than 40 parts per million (0.004%).

Lead. Not more than 10 parts per million (0.001%).

Loss on drying. Not more than 4%.

4. Tests

Assay. Boil about 1 gram, accurately weighed, with 50 ml of 0.1 N sulfuric acid for 10 minutes, or until the fatty acid layer

is clear, adding water if necessary to maintain the original volume. Cool, filter, and wash the filter and flask thoroughly with water until the last washing is not acid to litmus. Add methyl orange T.S., and titrate the excess sulfuric acid with 0.1 N sodium hydroxide. Each ml of 0.1 N sulfuric acid is equivalent to 2.015 mg of MgO.

Arsenic. Mix 1 gram of the sample with 10 ml of hydrochloric acid and 8 drops of bromine T.S., and heat on a steam bath until a transparent layer of melted fatty acid forms. Add 50 ml of water, boil down to about 25 ml, and filter while hot. Cool, neutralize with a 1 in 2 solution of sodium hydroxide, and dilute to 35 ml with water. This solution meets the requirements of the Arsenic Test.

Heavy metals. Place 750 mg of the sample in a porcelain dish, place 250 mg of the sample in a second dish for the control, and to each add 5 ml of a 1 in 4 solution of magnesium nitrate in alcohol. Cover the dishes with 7.6 cm short stem funnels so that the stems are straight up. Heat for 30 minutes on a hot plate at the low setting, then heat for 30 minutes at the medium setting, and cool. Remove the funnels, add 20 mcg of lead ion (Pb) to the control, and heat each dish over an Argand burner until most of the carbon is burned off. Cool, add 10 ml of nitric acid, and transfer the solutions into 250-ml beakers. Add 5 ml of 70% perchloric acid, evaporate to dryness, then add 2 ml of hydrochloric acid to the residues, and wash down the inside of the beakers with water. Evaporate carefully to dryness

again, swirling near the dry point to avoid spattering. Repeat the hydrochloric acid treatment, then cool, and dissolve the residues in about 10 ml of water. To each solution add 1 drop of phenolphthalein T.S. and sufficient sodium hydroxide T.S. until the solutions just turn pink, and then add diluted hydrochloric acid T.S. until the solutions become colorless. Add 1 ml of diluted acetic acid T.S. and a small amount of charcoal to each solution, and filter through Whatman No. 2, or equivalent, filter paper into 50-ml Nessler tubes. Wash with water, dilute to 40 ml, and add 10 ml of hydrogen sulfide T.S. to each tube. The color in the solution of the sample does not exceed that produced in the control.

Lead. Ignite 500 mg in a silica crucible in a muffle furnace at 475°-500° for 15-20 minutes. Cool, add 3 drops of nitric acid, evaporate over a low flame to dryness, and re-ignite at 475°-500° for 30 minutes. Dissolve the residue in 1 ml of a mixture of equal parts by volume of nitric acid and water, and wash into a separator with several successive portions of water. Add 3 ml of Ammonium Citrate Solution and 0.5 ml of Hydroxylamine Hydrochloride Solution, and make alkaline to phenol red T.S. with stronger ammonia T.S. Add 10 ml of Potassium Cyanide Solution. Immediately extract the solution with successive 5-ml portions of Dithizone Extraction Solution, draining off each extract into another separator, until the last portion of dithizone solution retains its green color. Shake the combined extracts for 30 seconds with 20 ml of dilute nitric acid (1 in 100), and discard the

chloroform layer. Add to the acid solution exactly 4 ml of Ammonia-Cyanide Solution and 2 drops of Hydroxylamine Hydrochloride Solution. Add 10 ml of Standard Dithizone Solution, and shake the mixture for 30 seconds. Filter the chloroform layer through an acid-washed filter paper into a Nessler Tube, and compare the color with that of a standard prepared as follows: to 20 ml of dilute nitric acid (1 in 100), add 5 mcg of lead ion (Pb), 4 ml of Ammonia-Cyanide Solution and 2 drops of Hydroxylamine Hydrochloride Solution, and shake for 30 seconds with 10 ml of Standard Dithizone Solution. Filter through an acid-washed filter paper into a Nessler tube. The color of the sample solution does not exceed that in the control.

Loss on drying. Dry at 105° to constant weight, using 2-hour increments of heating.

Packaging and storage. Store in well-closed containers.

Functional use in foods. Anticaking agent; binder; emulsifier.

VI. Description

A. General characteristics

Commercial preparations of magnesium stearate is a magnesium compound containing various proportions of stearic and palmitic acids. It occurs as a fine, white, bulky powder, having a faint characteristic odor.

B. Physical properties

Magnesium stearate is insoluble in water, alcohol and ether and is decomposed by dilute acids.

C. Stability in containers

Not available.

MAGNESIUM SULFATE

I. Nomenclature

A. Common names: Bitter salts, Epsom salts, Kieserite (naturally occuring mineral)

B. Chemical names: Magnesium sulfate, Sulfuric acid, magnesium salt, heptahydrate

C. Trade names: none

D. Chemical Abstracts Services Unique Registry Number: 10034998

II. Empirical Formula

$O_4MgS \cdot 7OH_2$

III. Structural Formula

$MgSO_4 \cdot 7H_2O$

IV. Molecular Weight: 246.47

V. Specifications

The Food Chemicals Codex, Second Edition (148), presents the following specifications for food grade magnesium sulfate:

1. Description

Small, colorless crystals, usually needle-like, with a cooling, saline, bitter taste. It is freely soluble in water, slowly soluble in glycerin, and sparingly soluble in alcohol. It effloresces in warm, dry air. Its solutions are neutral. A 1 in 20 solution gives positive tests for Magnesium, and for Sulfate.

2. Specifications

Assay. Not less than 99.5% of $MgSO_4$ after ignition.

Loss on ignition. Between 40 and 52%.

Limits of Impurities

Arsenic (as As). Not more than 3 parts per million (0.0003%).

Heavy metals (as Pb). Not more than 10 parts per million (0.001%).

Selenium. Not more than 30 parts per million (0.003%).

3. Tests

Assay. Weigh accurately about 500 mg of the residue obtained in the test for Loss on ignition, dissolve it in a mixture of 50 ml of water and 1 ml of hydrochloric acid, dilute to 100 ml with water, and mix. Transfer 50 ml of this solution into a 250-ml Erlenmeyer flask, add 10 ml of ammonia-ammonium chloride buffer T.S. and 12 drops of eriochrome black T.S., and titrate with 0.1 M disodium ethylenediaminetetraacetate until the wine-red color changes to pure blue. Each ml of 0.1 M disodium ethylenediaminetetraacetate is equivalent to 12.04 mg of $MgSO_4$.

Loss on ignition. Weigh accurately about 1 gram in a crucible, heat at 105° for 2 hours, then ignite in a muffle furnace at 450° ± 25° to constant weight.

Arsenic. A solution of 1 gram in 10 ml of water meets the requirements of the Arsenic Test.

Heavy metals. A solution of 2 grams in 25 ml of water meets the requirements of the Heavy Metals Test, using 20 mcg of lead ion (Pb) in the control (Solution A).

Selenium. A solution of 2 grams in 40 ml of dilute hydrochloric acid (1 in 2) meets the requirements of the Selenium Limit Test.

Packaging and storage. Store in well-closed containers.

Functional use in foods. Nutrient; dietary supplement.

VI. Description

A. General characteristics

Magnesium sulfate, heptahydrate, occurs as small, colorless, usually needle-like crystals. It has a cooling, saline, bitter taste.

B. Physical properties

The density of magnesium sulfate, heptahydrate is 1.67. It loses approximately one H_2O (7%) on exposure to air at ordinary temperature; loses four molecules of H_2O (about 28%) at 70-80°; loses five molecules of H_2O (36.5%) at 100°; loses 6 molecules of H_2O at 120° and becomes anhydrous at 250°. Rapidly reabsorbs water on exposure to moist air. One gram dissolves in 0.8 ml of water, 0.2 ml of boiling water, slowly soluble in 1.1 ml of glycerin and is slightly soluble in alcohol. Aqueous solutions are neutral.

C. Stability in containers

Not available.

Note: Magnesium sulfate may also occur as the trihydrate, an odorless powder, which is used in the preparation of aperients (Morison's paste).

VII. Analytical Methods

1. Atomic absorption spectroscopy has been widely used as a sensitive method devoid of most interference problems for the detection and estimation of various metals. Applications of this method to the determination of magnesium in biological material have been presented by Dawson and Heaton (171), Horn and Latner (334), and Sunderman and Carrol (743), who compare atomic absorption spectroscopy to the Bohuon method (see below).

The major probable source of interference in magnesium determination in biological material arises from formation of sulfate and phosphate salts which may under some flame conditions be resistant to thermal molecular dissociation. A study of the precision and accuracy of the atomic absorption method (811) showed a mean value of a sample known to contain 0.954 ppm to be 0.956 $\pm$ 0.035 ppm with a coefficient of variation of 3.7% based on seventeen determinations. The accuracy was 100.3%.

Other workers have used atomic absorption spectroscopy for the determination of magnesium in biological materials (258,261 ,341 ,346 , and393) and special note is made of the work of Schroeder, Nason, and Tipton (461) who used atomic absorption spectroscopy in the magnesium analysis of foods and water.

2. The Magnon reagent, the sodium salt of 1-azo-2-hydroxy-3-(2,4-dimethoxycarboxyanilino)naphthalene-1-(2-hydroxybenzene-4-sulfonic acid), of Mann and Yoe has been used for the spectrophotometric determination of magnesium in various biological mediums. The reagent is eight times more sensitive than the titan yellow reagent, also used in magnesium determinations. C. Bohuon (089) has presented a technique for the Magnon determination of magnesium which requires 100 µl of plasma or cerebrospinal fluid, which gave an accuracy of near 100% (sic) based on recovery of known

amounts of magnesium. Rice and Lapara have modified the Bohuon method to require only 40 ul of sample with increased sensitivity, accuracy and precision.

A discussion of the optimum conditions for the Magnon spectrophotometric determination of magnesium is presented by Burcar, et al. (109).

Phosphate is the major interfering ion causing low results in determinations, but may be removed by precipitation with aluminum at pH 5.5-5.6, being careful to avoid adding large excesses of aluminum which may also interfere with the determination. Gluconate does not interfere as in determinations with titan yellow. Calcium interference which is somewhat inconsistent may be compensated for by adding a known quantity of calcium to a standard curve developed at the time of reaction. Interference due to protein may be compensated for by precipitation of protein with trichloroacetic acid or addition of protein to the standards.

3. Another spectrophotometric method for determining magnesium in biological fluids employs the dye Eriochrome Black T (EBT), 1-(1-hydroxy-2-naphthylazo)-6-nitro-2-naphthol-4-sulfonic acid, sodium salt. The reagent forms metallochromic complexes with several cations, particularly calcium, which may interfere. The response of the calcium to the reagent can be eliminated by incorporating barium-EGTA (the barium salt of ethylene glycol bis(β-aminoethyl ether)-N,N,N',N'-tetraacetic acid). The calcium displaces the barium which does not interfere in the EBT reaction. Interferences due to iron, copper and zinc can be overcome by addition of trace amounts of cyanide ion. Connerty and Briggs (150) compared results from serum magnesium analyses by the EBT spectrophotometric method with results obtained from atomic absorption spectroscopy and found no evidence of a systematic difference between the methods.

4. Other methods for the determination of magnesium include flame emission spectroscopy (762, and 790); spectrochemical methods (517 and 568); fluorimetric methods (048, 392, and 555); the titan yellow colorimetric method (037, 156, 219, 353, 477, and 794).

With respect to ease of operation and accuracy and precision of results, none of these above methods offer any significant advantages.

VIII. Occurrence

A. Plants

Magnesium, which is essential for life, is present in all plants. The magnesium 2+ ion is a required co-factor for many enzymes, particularly phosphohydrolases and phosphotransferases. In green leaf plants, magnesium 2+ functions as a coordinate ion, stabilizing the porphyrin ring structure of chlorophylls a and b. While it has long been believed that green leafy vegetables are a major source of magnesium, Schroeder, et al. (461) showed that this has been found true only on a caloric basis and not on a weight basis. In their analyses (by atomic absorption spectrophotometry), they obtained mean concentrations of 170 ppm in 9 green, leafy vegetables; 194 ppm in 8 root vegetables; 241 ppm in 3 legumes; and 184 ppm in 11 vinous or fleshy vegetables. Schroeder et al. further found that analyses made on white and green parts of the same plant showed 55-61% more magnesium in the white portion on a weight basis. (For a complete breakdown of magnesium concentrations in various food plants, consult Table 22, in Biological Data, Section VI.)

B. Animals

Iain MacIntyre (463) has presented comparative magnesium concentrations for various animals. These values are shown in Table I. (Note:

Table I. Magnesium Content of Tissue and Serum of Various Animals (463)

Animal	Habitat	Tissue (mEq/kg)	Serum (mEq/l)
Man	T	19	1.7
Pig	T	22	
Dog	T	19	1.9
Frog	A	19	
Pike (teleost)	FW	26	
Cod (teleost)	M		5
Dogfish (elasmobranch)	M		12
Crab (limulus)	M		83
Lobster (Homarus)	M		13
Crab (Astacus)	FW		5
Octopod (Eledone)	M	25	114
Mussel (Anodonta)	FW	5	0.7

T = Terrestial

A = Amphibious

M = Marine

FW = Fresh water

These values were collected from sources dated between 1896 and 1950, and therefore, because of less precise methods of analysis, may not be accurate.)

The adult human body contains between 21 and 28 grams (approximately 2000 mEq) of magnesium (809). About half of this amount is contained in bone, while the remainder is distributed evenly between muscle and non-muscular soft tissue. Reported values of serum magnesium have been in the range of 1.4-2.5 mEq/l. (This range may result partially from the imprecise methods of analysis.)

Tables 2 and 3 show values as reported by Schroeder, et al. (677) for magnesium in human tissue (analyzed by flame emission spectroscopy).

C. Synthetics

No information available.

D. Natural Inorganic Sources

Magnesium, which makes up about 2.1% of the earth's crust, occurs as a constituent of several minerals. These minerals consist of two types: The solubles; magnesite ($MgCO_3$), dolomite ($MgCO_3 \cdot CaCO_3$), and meerschaum, serpentine, talc, and asbestos which are all silicates and the insolubles; carnallite, kainite, schoenite and kieserite.

Magnesium salts are present in hard water, the degree of hardness of the water depending largely on the concentration of these salts plus calcium salts. The median magnesium concentrations of the water supplies of the 100 largest American cities is given as 6.25 ppm (677), with the breakdown in various waters shown in Table 4

Table 2. Magnesium In Human Tissues, U.S. Values, 150 Accidental Deaths, Mean Values (677)

Organ	Organ weight (g)	Magnesium	
		Content (mg)	Concentration (μg/g wet wt ±S.E.M.)
Muscle	28,000	5300	189±7.2
Fat	12,500	300	24±0.2
Bone	10,000	11,000	1100±25.2
Blood	5500	210	38±0.4
Skin	4900	290	59±4.0
Connective tissue	2000	300*	150*
Liver	1800	310	172±5.2
Brain	1400	210	150±3.4
G.I. tract	1200	150	125±9.6
Lungs	1000	71	71±2.2
Heart	350	63	180±5.5
Kidneys	310	40	129±3.3
Spleen	180	23	128±4.2
Pancreas	100	16	160±4.4
Other	760	114*	150*
Total	70000	18397	285
Soft tissues	60000	9000	150

*Estimated from mean of all soft tissues. Analyses not done.
Note: Concentrations were obtained from analyses of 150 accidental deaths. Contents are based on organ weights of "standard man", using these concentrations. These 150 cases were selected from the total number analyzed on the basis of their dying accidentally and not of a known disease.

Table 3. Magnesium In Other Human Tissues, Mean Values (677)

	No. Samples	Ash (% of wet weight)	Magnesium	
			g/100 g ash (±S.E.M.)	μg/g wet weight (±S.E.M.)
Adrenal	10	0.5	0.89±0.10	45±5.0
Aorta	94	1.7	1.5 ±0.06	255±10.2
Diaphragm	91	0.98	1.7 ±0.05	167±4.9
Esophagus	57	0.89	1.4 ±0.06	125±5.3
Stomach	110	0.82	1.5 ±0.04	123±3.3
Duodenum	60	0.81	1.3 ±0.04	105±3.2
Jejunum	83	0.92	1.3 ±0.06	120±5.5
Ileum	77	0.75	2.1 ±0.12	158±9.0
Cecum	31	0.67	3.1 ±0.25	208±16.8
Sigmoid colon	88	0.68	2.6 ±0.22	177±15.0
Rectum	41	0.85	2.6 ±0.19	221±16.1
Omentum	72	0.25	1.2 ±0.06	30±1.5
Larynx	49	3.3	1.2 ±0.06	396±19.8
Trachea	57	1.9	1.2 ±0.05	228±9.5
Thyroid	14	1.4	0.98±0.19	137±26.6
Prostate	40	1.2	1.6 ±0.05	192±7.2
Testis	68	1.1	1.1 ±0.04	121±4.4
Ovary	16	1.0	0.94±0.06	94±5.9
Uterus	30	1.0	1.1 ±0.06	110±6.0
Bladder	98	0.78	1.1 ±0.05	109±3.9

Table 4. **Magnesium In Some Waters** (677)

	ppm
Natural waters	
Sea water, Caribbean	1300
Lake George, N.Y.	2.0
Lake Spofford, N.H.	8.4
Connecticut, River, Brattleboro, Vt.	23.0
Springs	
Brattleboro, Vt.	2.4
West Brattleboro, Vt.	0.8
Silverdale, N.H.	14.1
Chesterfield, N.H.	3.6
Saratoga, N.Y.(State Seal water)	5.3
Well, Putney, Vt.	5.2
Municipal waters	
Bangor, Penn. (Roseto)	0.8
Brattleboro, Vt.	1.4
Bridgeport, Conn.	3.8
Gibsonville, Ohio	16.4
Peterborough, N.H.	1.3
White Pains, N.Y	1.7

BIOLOGICAL SECTION

I. Acute Toxicity

A. Aquatic Animals

Dowden and Bennett (198) studied the median tolerance limits (TL_m) of magnesium sulfate ($MgSO_4$) and magnesium chloride ($MgCl_2$) with 3 species of aquatic animals: water fleas, Daphnia magna; a fish, the bluegill, Lepomis macrochirus; and snail eggs, Lymnaea sp. in two different support media; standard reference water and University Lake water filtered through glass wool. The results are shown on the acute toxicity Table 5 . The values given for the snail eggs represent the concentration of the magnesium salt at which 50 percent of the eggs hatched.

The authors found that increasing or decreasing the pH of a concentration of a chemical can cause an increase or decrease in its toxicity.

B. Mice

Barbour and Winter (037) studied the acute toxicity of magnesium chloride when administered subcutaneously to white mice (sex and age not given). The observations are shown in Table 6 .

C. Rats

1. Selisko and Ackermann (681) showed that intraperitoneal injections of high doses of magnesium salts (9.75 mg) produced symptoms of tetany in albino rats (inbred, 95 $\pm$ 5 g). The salts, $MgSO_4$ and $MgCl_2$ were administered in 3 concentrations; 1.5 ml with 6.5 mg Mg/ml, 0.75 ml with 13.0 mg Mg/ml, and 0.55 ml with 17.8 mg Mg/ml.

Many animals developed convulsions and all, including those that survived, developed narcosis. (See Table 7 for results.)

Table 5

Acute Toxicity of $MgSO_4$ and $MgCl_2$ in Aquatic Animals (198)

Substance	Animal	Route	Dosage mg/kg body wt.	Measurement
Magnesium chloride	Water fleas Daphnia magna	In standard reference water support medium	3,391 at 25 hr. 3,699 at 50 hr. 3,484 at 100 hr.	TL_m (mg/1)
Magnesium sulfate	Water fleas Daphnia magna	In glass wool filtered University Lake water support medium	963 at 24 hr. 929 at 48 hr. 861 at 72 hr. 788 at 96 hr.	TL_m (mg/1)
	Water fleas Daphnia magna	In standard reference water support medium	3,803 at 96 hr.	TL_m (mg/1)
	Fish Lepomis macrochirus	In standard reference water support medium	19,000 at 24 hr.	TL_m (mg/1)
	Snail eggs Lymnaea sp.	In glass wool filtered University Lake water support medium	10,530 at 24 hr. 6,525 at 48 hr. 6,300 at 72 hr. 6,250 at 96 hr.	TL_m (mg/1)

• Table 6. Acute Toxicity (037)

$MgCl_2$ (1% solution) mg/kg BW	No. of mice Survived	Died
1000	2	0
1050	1	3
1100	0	4
1200	0	2
1300	0	2
1400	0	2
1500	0	2
2000	0	2

Table 7. Acute Toxicity (681)

Magnesium salt	Conc.	No. Animals	No. with convulsions	Death
Chloride	a	30	11 = 37%	1 = 3%
Chloride	b	30	30 = 100%	25 = 83%
Chloride	c	30	30 = 100%	30 = 100%
Sulfate	a	40	3 = 7%	0 = 0%
Sulfate	b	30	22 = 73%	3 = 10%
Sulfate	c	30	18 = 60%	2 = 7%

Mg dose, 9.75 mg each: (a) 1.5 ml with 6.5 mg Mg/ml, (b) 0.75 ml with 13.0 mg Mg/ml, (c) 0.55 ml with 17.8 mg Mg/ml.

The authors observed that the intensity of convulsions was greater with salts of monobasic than with salts of dibasic acids. Also as Table 7 shows the toxicity of the chloride increased with increased concentration while with the sulfate, the medium concentration was most toxic.

The authors concluded that when aqueous solutions of magnesium salts are administered intraperitoneally, the toxicity is largely dependent on both the anion and the concentration.

2. The same authors, Selisko and Ackermann (682) compared the LD_{50}'s of several magnesium salts on Wistar albino rats (40 animals per salt, both sexes, 100 $\pm$ 5 g in weight). The salts were given in a single intraperitoneal injection. The corresponding concentrations of the doses given (0.1 to 0.25 ml) had the same magnesium content. The LD_{50}'s for magnesium sulfate and magnesium chloride at the three concentrations of Mg tested are shown on Table 8 .

D. Rabbits

Barbour and Taylor (039) subcutaneously injected normal, healthy adult rabbits with magnesium chloride. They found that 60% of the animals given a dose of 725 mg/g BW died. All of the ten animals given this dosage showed deep narcosis and six died, while of 5 rabbits given 750 mg/kg BW four showed deep narcosis and three died. Of eleven rabbits given 700 mg/kg BW, only five showed light narcosis and one deep narcosis with death. The figure of 725 mg/kg BW was therefore taken as the medium lethal dose for rabbits.

Table 8

Acute Toxicity of $MgSO_4$ and $MgCl_2$ in Mice, Rats and Rabbits

Substance	Animal	Sex & No.	Route	Dosage mg/kg body wt.	Measurement	Ref. Bibliogr. No.
Magnesium sulfate	Wistar albino rats	M & F 40	i.p.	150 mg Mg conc. of 6.5 mg/ml	LD_{50}	Selisko and Ackerman (682)
	Wistar albino rats	M & F 40	i.p.	130 mg Mg at conc. of 13.0 mg/ml	LD_{50}	Selisko and Ackerman (682)
	Wistar albino rats	M & F 40	i.p.	140 mg Mg at conc. of 17.8 mg/ml	LD_{50}	Selisko and Ackerman (682)
Magnesium chloride	White mice	Sex not given, 4	s.c.	1050	LD_{50}	Barbour and Winter (037)
Magnesium chloride	Albino rats	Sex not given, 30	i.p.	100 mg Mg	LD_{100}	Selisko and Ackerman (681)
Magnesium chloride	Wistar albino rats	M & F 40	i.p.	100 mg Mg at conc. of 6.5 mg/ml	LD_{50}	Selisko and Ackerman (682)
	Wistar albino rats	M & F 40	i.p.	90 mg Mg at conc. of 13.0 mg/ml	LD_{50}	Selisko and Ackerman (682)
	Wistar albino rats	M & F 40	i.p.	66 mg Mg at conc. of 17.8 mg/ml	LD_{50}	Selisko and Ackerman (682)
Magnesium chloride	Rabbits	Sex not given, 10	s.c.	725	LD_{50}	Barbour and Taylor (039)

II. Short-Term Toxicity

A. Rats

Moinuddin and Lee (515) studied whether feeding hydrogogue cathartics such as magnesium sulfate, which exert considerable osmotic effects in the lumen, would increase gastrointestinal growth in the rat.

Three feeding experiments were carried out for a 4-week period, with weanling male Sprague-Dawley rats (6 rats per group). In each experiment varying amounts of additional magnesium sulfate (anhydrous powder) replaced the cornstarch in a basal diet which already contained a salt mixture (3.8% anhydrous magnesium sulfate plus 0.02% manganous sulfate monohydrate).

Experiment a: 0.88 mmole/kg feed of $MgSO_4$ was added and fed at this level throughout the experiment. No unusual effects were observed at this low level of feeding.

Experiment b: The $MgSO_4$ was added initially at 8.64 mmole/kg feed and increased as follows: doubled on 9th day to 17.28 mmole/kg; redoubled on the 17th day to 34.56 and again on the 25th day to 69.12 where it was maintained for the remaining days of the experiment.

Experiment c: The $MgSO_4$ was added and maintained at 138 mmole/kg of feed throughout the experiment.

Controls: Basal diet with original salt mixture and cornstarch instead of added salt.

The observations of the dietary effect of $MgSO_4$ were:

(a) Chronic diarrhea.

(b) Depressed intake of food, also found when $MgCl_2$ is fed at excessive levels.

(c) At the excessive dose fed in experiment c, there was a large increase in the weight of the cecum which with the rectum was also noticeably increased in length.

(d) In experiment b, cecal contents were runny. In experiment c, both cecal and colon contents were runny.

(e) Serum alkaline phosphatase activity was higher in $MgSO_4$ fed rats than controls.

The authors note that the diarrhea and cecal distention observed in experiment c also has been reported to occur when $MgCl_2$ or $MgCO_3$ are fed to rats in large quantity.

B. Humans

1. A number of cases have been reported in the literature over the last forty years, describing magnesium sulfate poisoning. Several of these are summarized below chronologically.

(a) Thatcher in 1928 (766) reported the case of a 26 year old hospitalized male who died one hour after accidentally ingesting a large amount of magnesium sulfate solution (quantity or concentration not stated). The most significant gross pathological changes were a large quantity (approximately 1 liter) of fluid in the stomach and the dark red, hemorrhagic appearance of the stomach lining. The most marked microscopic pathological changes were small hemorrhages on the mucosa of the stomach and intestine. The amount of magnesium sulfate recovered in the stomach (57 g) was more than three times the normal dosage (15.5 g) given. The lungs were markedly congested. The author suggested that considering other reports

of toxicity and death resulting from magnesium sulfate administration, there are individuals for whom even an average dose may be toxic.

(b) Roller in 1936 (636) described a case in which a young woman (21 years) developed a picture of tetany with respiratory distress following injection of 2 ml of a 20% magnesium sulfate solution. The symptoms disappeared after a calcium salt solution was injected intravenously.

(c) Byron in 1939 (113) reported the cases of 5 young Tamil children with helminthiasis (age range 2-1/2 to 10 years) who died after the oral administration of a magnesium sulfate purge (mistura alba). All the deaths were characterised by the rapidity with which they occurred following the magnesium sulfate ingestion. The only postmortem abnormality noted was general congestion. The blood serum of one of the dead children contained 8 mg/100 ml serum as contrasted to the normal level of 2-3 mg/100 ml. The authors speculate that greater magnesium absorption than ordinarily occurs can happen in certain cases and particularly with strong solutions.

(d) Collins and Russell in 1949 (146) described a fatality owing to magnesium poisoning resulting from absorption of magnesium sulfate by the intestine, in a 4-year-old boy with primary megacolon. At autopsy the blood serum contained 30 mg Mg/100 ml. The authors considered the possibility that under certain conditions magnesium sulfate (Epsom salt) may become poisonous owing to its unexpected absorption. They note in

this regard that the gastrointestinal membrane is quite permeable to magnesium salts.

(e) Stevens and Wolff (735) in 1950 reported two cases of toxicity, one of which was fatal when a 300 ml solution containing 189 g of magnesium sulfate was administered rectally over a 3 hour period by slow Murphy drip. These toxic reactions resulted from significant absorption of the salt from the rectum which produced depressant cerebral effects. The authors observed that normal kidneys may not remove the magnesium sulfate from the blood stream fast enough to prevent amounts toxic to the central nervous system from accumulating.

(f) Rosler in 1952 (641) mentioned the case of one person (out of 12) treated for tapeworm with 80-120 g epsom salts (magnesium sulfate) dissolved in 200 ml water intubated into the duodenum, who died of central respiratory paralysis and magnesium narcosis. The author also mentioned being informed of another fatality resulting from curing tapeworm with magnesium sulfate. He pointed out that there are physiological conditions under which absorption of magnesium sulfate from the intestinal tract occurs. As a result the system is inundated with a large amount of magnesium leading to deep narcosis and paralysis of the respiratory center. He warned against the peroral use of large doses of epsom salts.

(g) In 1969, Colomb et al., (147) described the case of a young man who developed caustic lesions on the interior and exterior surfaces of his cheeks and lips, when epsom salts which was used as smelling salts spilled on his face.

(h) Rees (620) in 1970 reported the case of a pregnant female (aged 30) who apparently died from the effects of epsom salts which had entered the blood stream via the uterus in an attempted abortion. Abnormally high concentrations of Mg^{2+} ion were found in the heart blood (14.4 mg%) and in the peripheral blood (11.1 mg%). Prior to death, the woman allegedly reported a sensation of great heat passing through her body. This physiological phenomenon had been previously reported in connection with intravenous injections of a 42% solution of magnesium sulfate to volunteers.

(i) Willner (827) reported in 1971 on the death of a woman accidentally administered magnesium sulfate. The woman (59 years old) was a hospital patient who in the course of diagnostic tests was given 400 ml of 40% magnesium sulfate by duodenal tube. She died 6 hours later. On autopsy it was found that the blood magnesium level was about 33%. The author compared this with other reported blood magnesium levels following death from magnesium poisoning and concluded that magnesium levels about 15% indicated a toxic content. He also considered his finding of 8-10% magnesium in the vitreous fluid as probably 2 to 3 times higher than is normally present.

2. Allison (012) discussed a toxic sulfate effect from water found in about 1/4 the state of Minnesota which is high in $MgSO_4$ (as well as other sulfates). In the regions in which this high sulfate containing water (770 ppm to 3,590 ppm) occurs both man and livestock suffer from its drastic cathartic action and weakening effect. Calves are stunted in growth and many of the cattle die prematurely. Cattle from Dakota and Montana where similar water exists have been observed to be similarly affected. The author suggested that people may be affected in the same way but perhaps to a different degree. He urged a continuation of experimental studies in particular those concerning the effect on the equilibrium of calcium in the body.

3. Hirschfelder (323) studied the effects of oral administration of magnesium sulfate. Normal persons showed no rise in the level of magnesium in the blood plasma after ingesting epsom salts and thus had no ill effects. Patients with renal disease had entirely different results. When one or more purgative doses (25 g per dose) was administered orally there was a large rise in plasma magnesium within 4 to 6 hours. Many of the patients showed a syndrome of high plasma magnesium (hypermagnesemia, 9-11 mg/100 ml plasma) which was accompanied by somnolence or light coma. The author believed that epsom salts purgation in nephritic patients can induce a magnesium coma which is easily mistaken for uremic coma. The difference is significant because magnesium coma can be treated by intravenous injection of calcium chloride.

There is another condition, hypomagnesemia or low plasma magnesium characterised by convulsions or muscular twitching. This condition is relieved by oral administration of epsom salts. The author believed

that hypomagnesemia with its accompanying condition of hyperirritability of the neuromuscular system is probably more common than is realized.

4. Abramowitz and Russo (001) described the case of a "fixed" eruption caused by magnesium hydroxide. The patient had previously suffered cutaneous lesions and a rash on her hands after ingesting phenolphthalein and phenobarbital (0.06 g). Subsequently on taking either magnesium hydroxide tablets or magnesia magma (suspension of magnesium hydroxide in water) the eruption recurred.

5. Randall et al. (616) studied the relation of clinical manifestations of hypermagnesemia and serum magnesium level. Marked hypermagnesemia and severe manifestations of toxicity were developed by five patients with renal disease who were administered prescribed magnesium medications. (Table 9 summarizes the results.) Fourteen patients with chronic renal failure ingested 180 ml daily (6 equal 30 ml doses at 3 hour intervals on 3 consecutive days) of one of two magnesium antacids (either the hydroxide or the trisilicate). (The results are summarized on Table 10). Similar doses under the same circumstances were given to six normal subjects. (The results are summarized on Table 11 .)

Table 9. Clinical and Laboratory Data in Patients With Toxic Manifestations of Hypermagnesemia (616)

Patient No.	Diagnosis	Creatinine Clearance ml/min	Hospital Day	Magnesium Intake: Oral	Magnesium Intake: Parenteral	Serum: Mg mEq/l	Serum: Ca mg/100 ml	Serum: PO_4 mg/100 ml	Serum: Na mEq/l	Serum: K mEq/l
			5	$Mg(OH)_2$[c]	$MgSO_4$ 40 mEq im[d]			2.1	145	2.6
1	Bleeding duodenal ulcer	12	6		$MgSO_4$ 16 mEq iv[d]	7.5	5.1	2.6	148	3.7
			3-7		$MgSO_4$ 116 mEq im[d] in 5 days		6.7	5.0	126	3.2
			13			1.4			132	5.4
2	Acute pyelonephritis with papillary necrosis	30	14-16		$MgSO_4$ 220 mEq im[d] in 3 days	7.2	7.2	5.0	138	5.0
			21			3.0	7.4		138	5.0
		10	6	$MgSO_4$ (laxative)			6.8	9.6	122	3.8
3	Malignant hypertension	<1	10			4.2				7.3
			15			5.4	6.0		140	8.4
			1	$Mg(OH)_2$[c]					135	3.3
4	Cirrhosis with oliguria	20	5			9.1		4.3	120	5.5
		10	6			7.6	6.6	3.4	121	5.7
			1	$Mg(OH)_2$[c]			8.9	7.4	138	5.3
5	Diabetic glomerulosclerosis	7	10			5.0	8.9			7.1
			14			5.4	7.6	7.9	130	8.4

[a]NPN rather than BUN.
[b]All interval measurements are in seconds.
[c]Given as Maalox, 120-300 ml daily.
[d]Administered as 50% $MgSO_4 \cdot 7H_2O$ as prepared by Lilly and Co.
[e]Arterial pCO_2 39 mm Hg at this time.

Table 9 (Con't)

Patient No.	Serum Cl mEq/liter	Serum CO_4 mM/liter	Blood Creatinine mg/100 ml	Blood pH (art.)	Blood Urea N[a] mg/100 ml	Hematocrit Reading %	Urine ml/day	Electrocardiogram[b]	Toxicity
1	85	26			62[a]	43	>1,000	PR, A6, QRS .09, QT_c .49, T waves flat	Following injections: coma, areflexia, apnea, hypotension
	80	41	7.0	7.49	100[a]	48			Apnea and death after injection
2	84	14			60	26		Normal	First course of magnesium reduced symptomatology presumably related to magnesium depletion
	112	17			36				
	106	15	3.2		38		>1,000	Nodal rhythm, QRS .16, QT_c .47, T waves ininverted	Second course of injections followed by arrhythmia, transient asystole, hypotension, coma, decreased respirations
	107	17			73	26		Normal	Improved as serum magnesium level fell
3	92	17	4.8		90[a]	29	200	PR .16, QRS .10	
							0		
	90	8	53		228[a]		0	PR .22, P waves flat, QRS .13, T waves tall	P-R prolongation. Coma and death probably related to severe uremia
4	73	26	4.0		81	30	300		
	60	36			130		100		Progressive coma, hypotension, diminished reflexes and respirations
	60	37[e]	6.6	7.59	135	36	50		
5	106	15	11.0		90	33		PR .15, QRS .08,	
	104	15			110		600	QT_c .41, PR .32, QRS .16, QT_c .50, ST depressed, T waves tall	Progressive drowsiness, confusion, hypotension, dysarthria, difficulty voiding, diminished respirations, P-R prolongation
	99	14	12.0		125	33	1,000	Same	Death

Table 10. Clinical and Laboratory Data in Patients with Renal Disease Ingesting Magnesium-antacids Experimentally

Patient No.	Diagnosis*	Creatinine Clearance	Medication†	Serum Magnesium		Blood Urea N	Serum			Urine Magnesium		Toxicity**	
				Before Antacid	After 3 days		Cr						
		ml/min		*mEq/liter*		*mg/100 ml*	*mg/100 ml*			*mg/24 hr*			
6	Chronic glomerulonephritis (b)	30	Maalox	2.0	3.0	36	3.2		4.0	65	170	Nausea	
7	Nephrosclerosis (a)		Maalox	1.8	4.3	116	9.0		12.3			Sleepy, malaise	M
8	Diabetic glomerulosclerosis (b)	11	Gelusil	2.1	3.4	106	7.0			84	136		
9	Chronic glomerulonephritis (b)	29	Maalox	2.8	3.6	75	5.2						
10	Diabetic glomerulosclerosis (b)	28	Maalox	2.4	3.0	62	2.4		5.3				
11	Diabetic glomerulosclerosis (c)	8	Maalox	2.4	2.6	150	11.0			65	82		
12	Chronic glomerulonephritis; congestive failure (c)	10	Maalox	2.3	3.9	108	5.6					Drowsy	
13	Nephrosclerosis; nephrocalcinosis (a)	8	Maalox	1.7	3.5	100	5.6						
14	Amyloidosis (a)		Maalox	2.0	2.7	40							
15	Nephrocalcinosis (Milk-alkali) (c)‡	55	Gelusil	2.2	2.5	37	1.5			125	300		
16	Nephrocalcinosis (Milk-alkali) (b)	30	Maalox	2.4	3.5	51	3.2						
17	Chronic pyelonephritis (a)	8	Maalox§	2.6	4.9	100	7.9			50	120	Nausea, drowsiness, malaise; difficult micturition and defecation; muscle twitches; postural hypotension, slight P-R prolongation	
18	Chronic glomerulonephritis (a)	4	Maalox	1.8	3.2	200	16.4			32	44	Drowsiness	
19	Adenocarcinoma prostate (c)		Maalox	1.8	3.6	60							

*Established by autopsy examination (a), renal biopsy (b), or clinically (c).
†See subscript Table 3.
**Toxic manifestations began during the period of medication and subsided upon stopping the medication as the serum magnesium concentration fell.
‡Reported in detail elsewhere (21).
§Similar results were obtained during the ingestion of a comparable amount of magnesium hydroxide as Milk of Magnesia (USP).

Table11. Serum and Urine Magnesium in Normal Subjects Ingesting Antacids (616)

Subject No.	Creatinine Clearance	Antacids*	Serum Magnesium		Urine Magnesium	
			Before Antacid	After 3 days	Before Antacid	Third Day
	ml/min	180 ml/day	mEq/liter		mg/24 hr.	
1	110	Maalox	1.9	2.1	122	234
2	120	Maalox	2.1	2.0	87	284
2	120	Gelusil	2.0	2.0	85	400
3	100	Maalox	2.2	2.3		
4	115	Maalox	1.9	2.0		
5	130	Maalox	1.7	1.8	115	270
6	140	Gelusil	2.3	2.5	125	306

*Provides approximately 2.8 g (235 mEq) of magnesium as $Mg(OH)_2$ in Maalox and 2.9 g (240 mEq) of magnesium as magnesium trisilicate in Gelusil.

The authors pointed to several observations for particular emphasis:

(a) The serum concentrations at which toxic manifestations appeared in patients with renal disease were much lower than those in the normal subjects.

(b) Patients with renal failure have a limited capacity to excrete magnesium.

(c) The potential danger of magnesium absorbed from ingested laxatives and antacids in patients with renal disease has not been fully appreciated.

(d) Patients with severe renal failure who ingest customary doses of standard laxative and antacid preparations, may develop significant deviations of serum magnesium accompanied by manifestations of toxicity within 3 days.

6. Brady and Williams (098) reported the case of a premature infant with hypermagnesemia. The mother was administered magnesium sulfate (a total of 11 gm) prior to the infant's birth. The infant had a high serum magnesium level (15 meq/l at 9 and 16 hours and 9.8 meq/l at 24 hours) and showed severe symptoms of motor and respiratory paralysis. The condition was cured by exchange transfusion. The authors concluded that the magnesium sulfate therapy given to the mother had profound effects on the fetus.

7. Lipsitz and English (448) reported on sixteen cases of hypermagnesemia in newborn infants. All of these infants were born to mothers given magnesium sulfate for toxemia. In both the maternal and fetal serums, magnesium levels were higher than expected. The clinical

manifestations of the newborn were similar to those of adults with hypermagnesemia. The authors urged awareness of this problem.

8. Stone and Pritchard (736) on the other hand reported that they had not found any deleterious effects from magnesium sulfate administered to mothers prior to delivery. They claimed that over a 14 year period, 7000 infants were born of mothers who had received magnesium sulfate therapy. They further claimed that even though the serum level of magnesium in the fetus rapidly approached that of the mother, it could not be correlated with any ill effect. The authors specifically cited the previously reported study of Lipsitz and English.

9. Lipsitz (447) warned again that more detailed study has shown that if continuous intravenous infusion of magnesium sulfate is given to the mother for more than 24 hours, the newborn will manifest all the signs of hypermagnesemia. Two routes of magnesium sulfate administration were studied (27 mothers, intravenously; 8 mothers, intramuscularly after initial intravenous injections). The author concluded that when toxemic mothers are administered a large dose of magnesium sulfate or prolonged magnesium sulfate therapy, there is great likelihood that their infants will manifest the symptoms of magnesium toxicity. These symptoms which are similar to those described in adults (see Randall et al. 616, Biology Section II, B, 5) with hypermagnesemia are unlike those born to toxemic mothers without magnesium sulfate therapy. Magnesium toxicity should be recognized and treated in such newborns.

III. Long-Term Toxicity

No information available.

IV. Special Studies

A. Frogs

Chiray et al. (138) studied the effect of magnesium chloride on the frog intestine in vivo. A cannula was introduced via a lateral opening in frogs with previously destroyed medullas (number and sex not given). By making a second opening 2 or 3 cm away to allow the perfusion liquid to exit, the first 2 or 3 cm of the small intestine could be studied. In the first part of the experiment Tyrode's solution at 38° was perfused and the number of drops passing the section of intestine being studied during one or more minutes was counted by means of an electric drop counter. The perfusion was carried out at a constant pressure and temperature (10-15 cm of water at 38°). Then the Tyrode's solution was replaced by water containing magnesium chloride (from Chatel-Guyon). In a few minutes there was an approximate 70 percent decrease in the drop flow from 50-55 per minute to about 15. This reduced flow which was established in a few minutes remained as long as the perfusate contained magnesium chloride. When this was replaced by physiological solution, the flow returned to its former rate after a few drops.

The authors relate this effect, which they ascribe to a strong amplification of normal peristaltic waves, to what happens when magnesium chloride exerts a laxative effect in man.

They point out that the large doses (1500 cc and up daily) which were previously administered therapeutically at Chatel-Guyon have been discontinued because they cause diarrhea. Present dosages of a few hundred

grams which are used to regulate intestinal function apparently play a decontracting role in the case of spasmodics and a contracting role with atonics. The authors suggest it would be of interest to study the effect of magnesium chloride containing water on an intestine contracted by various pharmacological agents.

B. Mice

1. Labkovsky (424) studied the effect on the induction of pulmonary adenomas with urethan in mice, when magnesium salts (chloride or sulfate) are administered orally or subcutaneously. All the experiments were carried out on stick-bred white mice (19-20 g, number and sex not given).

The six oral experiments, each with its own controls were as follows:

(a) Starting 2 weeks prior to first urethan injection (1 g/kg in 5% solution administered intraperitoneally twice per day every 4 days), $MgCl_2$ (320 mg/kg) was given in drinking water for 10 weeks.

(b) The same amount of $MgCl_2$ was administered via stomach tube. Administration started one day prior to first urethan injection (same as 1) and continued for 10 days.

(c) As above.

(d) As above.

(e) Everything the same as in 2, 3, and 4 except $MgSO_4$ administered.

(f) Everything the same as in 2, 3, 4, and 5 above except $MgSO_4$ administered subcutaneously.

The results showed the following:

Experiment	Percent when average no. of pulmonary adenomas per control mouse taken as 100%
a	135
b	216
c	215
d	112
e	194 and 196
f	173

In another series of four experiments $MgSO_4$ was injected subcutaneously daily (5 ml/kg of 0.5% solution) for 10-15 days. These experiments were as follows:

(1) Urethan (same amount as above) administered 2 days after cessation of $MgSO_4$ injections.

(2) $MgSO_4$ injections started 10 days after second urethan injection.

(3) $MgSO_4$ injections started 2.5 months after urethan administration.

(4) Control.

The results were:

Experiment	Percent when average no. of pulmonary adenomas per control mouse taken as 100%
a	174
b	146
c	114

The author concluded:

(1) Magnesium salts (chloride or sulfate) stimulated the induction of pulmonary adenomas by urethan in mice.

(2) The method of administration or the composition of the salt (chloride or sulfate) did not have any effect.

2. Bazikyan and Akimov (051) studied the antineoplastic effect of magnesium chloride in mice under experimental conditions. A total of 259 mature male CC57BR mice were used in all the experiments described below. Skin papillomas were induced by rubbing a 0.1% benzene solution of 9,10-dimethyl-1,2-benzanthracene (DMBA, 50 mg/kg per mouse) on the posterior portion of the spine on alternate days for one month (15 applications). Two series of experiments were carried out as described below:

(a) In series 1, there were 2 subgroups. In one the mice were given drinking water to which magnesium chloride (60 mg/kg/day) had been added throughout the entire time the carcinogen was applied (30 days). The other group which were controls received ordinary drinking water. (See Table 12 for details and results.)

(b) In series 2, there were 4 subgroups. One was a control group similar to the one in series 1 above. Subgroup 1 received $MgCl_2$ (60 mg/kg/day) as above 7 days prophylactically and 30 days with DMBA. Subgroup 2, received $MgCl_2$ as in subgroup 1 but with DMBA for 53 days. Subgroup 3 received the $MgCl_2$ from the eighth day after the first application of DMBA and for 48 days thereafter, when the first papilloma appeared. See Table 12 for details and results.

Some observations were:

(a) Magnesium chloride showed definite antitoxic action in series 2 subgroup 2 in particular, in which $MgCl_2$ was administered for the longest time. (See Table .)

Table 12 a. Effect of Magnesium Chloride on the Origin of Skin Papillomas in Mice (051)

Series of experiments	Animal subgroups	No. of mice at start of expt.	No. of mice at end of expt.	No. of mice died %	p
I	Control	30	22	27	0, 5
	Experimental	30	24	20	
II	Control	29	19	35	
	Experimental 1	30	24	20	0, 3
	Experimental 2	31	27	10	0, 05
	Experimental 3	29	17	42	

Table 12 b.

Series of experiments	Animal subgroups	No. of mice with tumors abs.	No. of mice with tumors in %	p	Avg. no. of tumors per mouse abs.	Avg. no. of tumors per mouse % of control	p
I	Control	18	82	0, 5	1, 1 ± 0, 16	100	0, 1
	Experimental	16	66	0, 3	0, 80 ± 0, 11	73	
II	Control	14	74		0, 90 ± 0, 17	100	
	Experimental 1	12	50	0, 1	0, 63 ± 0, 12	70	0, 1
	Experimental 2	11	41	0, 05	0, 55 ± 0, 14	51	0, 07
	Experimental 3	11	65	0, 05	0, 82 ± 0, 14	90	0, 7

(b) On the other hand in subgroup 3 of series 2, 42% of the mice died. The authors postulated that this was due to the 8-day delay in administration of $MgCl_2$.

(c) In all the groups of both experimental series the first tumors appeared between days 48 and 54. Thereafter there was a different rate of increase.

(d) The experimental mice in series 1 had fewer tumors than the controls throughout the experiment.

(e) Subgroup 3 of series 2 had the largest number of animals of all that received magnesium, both with respect to skin papillomas and number of tumors per mouse. The death rate was also highest in this group. The authors conclude from this observation that: (a) magnesium chloride must be administered both for several days before topical application of the carcinogen and concomitantly with it, in order to manifest an antitoxic and antineoplastic action and, (b) that irreversible changes in the cells which later become malignant are produced from the very first application of carcinogen.

In another series of experiments the effect of magnesium chloride on connective tissue tumors was studied. The connective tissue sarcomas were induced in all 3 experimental groups by a single injection of 3 mg of 1,2,5,6-dibenzanthracene (DBA).

The controls received tap water throughout the duration of the experiment (200 days). Group 1 drank water with added magnesium chloride (60 mg/kg) for 30 days and group 2 for the duration of the experiment. (See Table 13 for details and results.)

Table 13. Effect of Magnesium Chloride on the Induction of Subcutaneous Sarcomas in Mice (051)

Groups of animals	No. of mice			p	No. of mice with tumors		p
	at start of expt.	at end of expt.	died %		abs.	in %	
Control	30	18	40	0, 3	15	83	
Experimental 1	19	15	21	0, 02	10	66	0, 2
Experimental 2	21	19	10	0, 05	7	37	0, 01

The observations were:

(a) The group with the highest mortality and the first animals to die from the toxic effects of DBA were the controls, as in the previous experiments.

(b) Also as in the previous experiments, the group with the lowest mortality had the smallest number of mice with tumors. The authors concluded from this experiment that the ingestion of $MgCl_2$ lowered the number of DBA induced sarcomas.

They concluded from all the above experiments that:

(a) Under the conditions of the experiments and in the amount administered, $MgCl_2$ reduced the toxicity of DMBA and DBA.

(b) Under their experimental conditions $MgCl_2$ markedly decreased the number of skin papillomas induced by DMBA and subcutaneous sarcomas induced by DBA.

C. Rats

Wies (826) reported on experiments to determine whether magnesium sulfate is teratogenic in rats. A series of experiments were carried out using 34 albino rats (18 female and 16 male, 120-200 days old). The females were given subcutaneous doses of sterile magnesium sulfate solution (0.8 mg/kg $MgSO_4 \cdot 7H_2O$) on alternate days (for approximately 8 weeks). The females were all mated at least once.

It was observed that:

(a) The fertility of females receiving magnesium sulfate was low (15 percent).

(b) Varying degrees of toxicity were produced by the magnesium sulfate including death.

(c) Defects in the brain and spinal cord of at least one fetus were observed. (It is not stated how many fetuses were produced nor in how many of those produced were the described defects observed.) Specifically the defects consisted of a diminution of the number of neuroblastic cells in a circumscribed area of the midbrain. In the spinal cord the lateral walls had not fused over the central canal leaving it open.

D. Guinea Pigs

Chiray et al. (138) carried out the same experiment with guinea pigs in which the action of magnesium chloride containing water on the intestine in vivo was studied as has already been described in detail with frogs, in A, 1 in this section.

Before inserting the cannula the guinea pigs (number and sex not given) were lightly anesthetized with ether. In the guinea pig experiment the section of intestine studied was the end of the ileum and the ileocecal valve. The original perfusion solution was not specifically specified but referred to as physiological perfusion liquid.

The main differences in the response of the guinea pig were that the rate of flow was less slowed down (from 38 drops per minutes to 28 as compared to 50-55 drops per minute to 15 in the frog) and return to the original rate of flow (with physiological solution) took longer to reestablish than with the frog. In all other respects the results with both species were identical. Perfusion of a small section of the small intestine with water containing magnesium chloride caused the flow of the perfusate to be slowed down owing to the contraction of the smooth

fibers of the intestine. This contraction was caused by a strong exaggeration of the normal peristaltic waves.

E. Rabbits

1. Serbescu (686) studied the influence of magnesium sulfate on tar-induced carcinomas. Two groups of 30 rabbits each were used (age and sex not given). One served as controls and the other was administered magnesium sulfate by stomach probe (20 mg/kg, dissolved in redistilled water) every day except Sunday. All the animals were painted on the inside of both ears with tar oil three times per week. Animals in both groups which died were immediately replaced, thus in the course of the experiment a total of 123 rabbits were used (75 experimental and 48 controls). Both pathological studies (the digestive tract and the ganglionic system) and histological tumor examination were carried out. The following were noted for the magnesium treated animals:

(a) A higher mortality rate.

(b) Cancer developed more easily.

In the same paper Mr. P. Delbet submitted a rebuttal which made essentially the following points:

(a) His own experience with the magnesium halogen salts has indicated a restraining effect on cancer development.

(b) Magnesium sulfate would not necessarily behave similarly for two reasons: (a) it is not as well absorbed as the chloride, (b) the sulfate tends to make the urine alkaline whereas the halogen salts reduce its pH. Delbet does not explain why he considers this significant.

(c) The described method of sulfate administration would probably weaken the animals both by acting as a purge and by preventing them from getting adequate nourishment.

(d) Delbet's experimental work in which he demonstrated a reduction of tumor formation when animals were injected with a mixture of magnesium halogen salts, was repeated successfully. The experimenter, Braier, additionally determined that the C/N ratio in the urine of cancerous rats was lowered in the controls but remained normal in the magnesium treated animals. Delbet concludes from this that the poor utilisation of fats and carbohydrates is a factor in cancerous cachexia and that large doses of magnesium halogen salts have a therapeutic effect.

2. Neal and Neal (537) studied the effect of hard water and water containing magnesium sulfate on atherosclerosis in rabbits. Four groups of New Zealand rabbits (5 per group, age and sex not given) were given drinking water as follows for 3 months:

Group I - distilled water

Group II - hard sulfur water

Group III - one tablespoon of calcium carbonate suspended in one gallon of distilled water

Group IV - 7 tablespoons of magnesium sulfate (Epsom salt) dissolved in one gallon of distilled water (30,000 ppm).

The diet of all groups was Purina rabbit pellets with 1% crystalline cholesterol and 5% butter. The aortas and hearts of all the rabbits were studied.

The following was observed:

(a) Group I rabbits (distilled water) had the most atherosclerosis.

(b) Group II and Group III rabbits had moderate atherosclerosis.

(c) Group IV rabbits (magnesium sulfate) had no atherosclerosis, did not have diarrhea and were larger and fatter (more subcutaneous fat) than the rest of the rabbits.

(d) Although the blood serums of rabbits from each group contained the same amount of fat, the serums from the Group IV rabbits (magnesium sulfate) were relatively clear while those from the other 3 groups were turbid or "milky".

The authors raised the questions of whether there is a protective substance in hard water or whether there is a specific effect of magnesium on fat metabolism. They suggested the necessity for further studies using other ionizing compounds.

F. Dogs

1. Hazard and Wurmser (308) studied the effect of magnesium chloride on the excitability of the sympathetic nervous system of dogs. Male dogs (6.6 kg, number not given) were administered $MgCl_2$ intravenously at various strengths (0.01 g/kg to more than 0.10 g/kg).

It was found that:

(a) Weak doses of $MgCl_2$ (0.01-0.05 g/kg) slightly increased the heart's response to excitation by accelerators.

(b) Strong doses (0.10 g/kg and up) had the opposite effect.

(c) In vagotomized or atropinized dogs large doses of $MgCl_2$ increased the accelerating effect of adrenaline and lessen the hypertensive action of nicotine.

(d) A depressant effect on the viscera, particularly the kidney, resulted from large $MgCl_2$ doses.

2. Maxwell et al. (490) studied the effects of hypermagnesemia on the hearts of intact dogs. Hypermagnesemia was induced in 6 dogs (13-30 kg, sex not given) by intravenous injection of a 10% solution of magnesium chloride. In an unstated number of other dogs 2 ml/kg of 10% magnesium chloride was administered by infusion. Before and after the induction of hypermagnesemia, cardiac output, coronary flow, and vascular pressures were measured. The main changes which took place were tachycardia and a fall in systemic pressure. The authors postulated that under the conditions of the experiments the resulting tachycardia and hypotension appeared to be associated with variation in the calcium-to-magnesium ratio owing to the increase in magnesium.

G. Humans

1. Bernstein and Simkins (076) studied the intravenous effects of therapeutic doses of magnesium sulfate on the heart. One hundred adult patients were used in the study, 34 apparently free of cardiovascular disease (but with various other diseases) and 66 with various forms and degrees of cardiac disease (see original paper for details). Warmed magnesium sulfate solution (10 cc of a 10% aqueous solution) was injected into a large antecubital vein of the patient. Three sets of electrocardiograph tracings (one from each lead) were made for each patient.

The authors considered that the most salient feature of their study was the lack of uniformity in the changes in the heart brought about by the magnesium sulfate injections. In a fairly high percentage of cases there were slight changes in QRS complexes and T waves indicating a direct effect on the

myocardium of the ventricles. The authors concluded that intravenous magnesium injections exerted no deleterious effect on the human heart.

2. Boyd and Scherf (092) studied the therapeutic use of magnesium sulfate in paroxysmal tachycardia. Eleven patients (10 cases of paroxysmal tachycardia and 1 case of flutter) were treated by intravenous injection of magnesium sulfate in concentrations of 10% or 20%. The results were favorable in 3 out of 8 cases for the 10% solution. The effect was immediate. It was more successful at the higher concentration (15 to 20 cc of a 20% solution); 8 out of 8 cases. The authors considered this procedure safe but cautioned against its use in the presence of: marked myocardial damage, marked intraventricular conduction, or gallop rhythm.

3. Ozsoylu (565) examined the effect of giving magnesium sulfate to control blood sugar levels in diabetic children. Three children (from 5-14 years) were given an intravenous infusion of a 3% magnesium sulfate solution (150 mg/kg). The blood sugar levels were determined on the three days prior to magnesium sulfate therapy as well as on the fourth day when the magnesium sulfate was given. The same amount of insulin was given on all four days. Three patients (controls) who did not have diabetes were given the same amount of magnesium sulfate on the fourth day following three days of checking their blood sugar levels. It was found that the blood sugar level of all the diabetic patients dropped considerably (116 mg%, 195 mg% and 234 mg%) within one hour following the infusion of magnesium sulfate. It appeared (see Table in original paper for details) that the higher the blood sugar level the greater effect the magnesium sulfate had on decreasing it. There was no effect on the normal blood sugar levels of the controls, from the magnesium sulfate infusion. The author expressed

the hope other investigators will be interested to gather further data since there are not too many diabetic children in Turkey.

4. Frewin et al. (237) attempted to clarify the vasodilator action of magnesium sulfate. Eight volunteer subjects (7 medical students and one patient with a sympathetically denervated forearm) were used. Intra-arterial infusions of magnesium sulfate (20 and 50 mg/min) were given into the brachial artery at the elbow of one side. Forearm or hand blood flow was measured.

The results of the experiments demonstrated that:

(a) Magnesium sulfate has a direct local vasodilator action on the blood vessels of the forearm.

(b) The vessels of both the skin and the muscle are affected.

(c) The local direct action on the blood vessels was further confirmed by the presence of a vasodilator action in the patient with the sympathetically denervated forearm.

(d) Administration of propanolol, hyoscine or mepyramine did not modify the response. This indicated that the dilator action is not caused by stimulation of β-receptors, a cholinergic mechanism or the release of histamine.

(e) The simultaneous administration of an equimolar dose of calcium gluconate, a vasoconstrictor, abolished the dilator action of magnesium sulfate.

BIOCHEMICAL ASPECTS

I. Breakdown

The breakdown of the various magnesium compounds in the body is discussed in the following sections. No breakdown in storage, processing, or cooking has been noted in current literature.

II. Absorption and Distribution

A. Rats

Alcock and MacIntyre (007) studied the interrelation of calcium and magnesium absorption by studying the effect of calcium deficiency on magnesium absorption in rats.

Female rats (28 animals, 90-110 g) were tube-fed diets deficient in calcium for 18 days. A control group (26 animals) received a diet (also by tube) with calcium present in a normal amount (see Tables 14 and 15 for details). Balance techniques were used to study the effect of dietary calcium deficiency on the absorption of magnesium. (See original paper for details of method.)

The excretion of magnesium by the calcium deficient animals was markedly diminished. There were no abnormal signs in these animals apart from the fact that the feces were soft and less bulky than normal. Also the fecal dry weight was diminished. The mean daily excretion of magnesium and calcium together with that of the normal control animals is shown below.

Table 14

The Electrolyte Content of the Diets (007)

Type of diet	Mg	Ca	K	Na	Phosphate
Normal	34	300	145	45	100
Ca deficient	34	3.0	145	45	100

(Magnesium, calcium, potassium and sodium content is expressed in terms of m-equiv/kg of diet; phosphate content is expressed in terms of m-moles/kg of diet.)

Table 15

The Daily Intake (μ-equiv/rat) of Calcium and Magnesium (007)

Diet	Magnesium	Calcium
Normal Control	272	2400
Calcium Deficient	272	2

Mean daily fecal excretion of calcium and magnesium (u-equivalents). Results are expressed as mean (number of determinations) ± S.E.M.

Excretion			
Magnesium		Calcium	
Experimental group	Normal group	Experimental group	Normal group
84.9(26) ± 6.4	154.0(28) ± 6.3	6.2(26) ± 0.95	1684(28) ± 57.4

The authors concluded that their results strongly suggested the existence of a common transport mechanism for the transfer of calcium and magnesium across the intestinal wall. They considered the increased magnesium absorption from the gut in calcium deficient diets as evidence in support of this mechanism. They suggested both that a similar mechanism may exist in the kidney and that the possibility exists that such a common absorptive mechanism for calcium and magnesium in both the intestine and renal tubule may exist in many different species.

B. Rabbits

Aikawa and Burns (004) investigated the maternal-to-fetal transfer of magnesium and its uptake by various maternal and fetal tissues in the rabbit. The radioactive isotope ^{28}Mg (as magnesium sulfate solution) used in the study was injected into the marginal vein of the ears of eleven pregnant domestic albino rabbits (between 28-30 days of gestation). A total of 86 fetuses and their placentas were removed from the rabbits at intervals ranging from 7 minutes to 26 hours.

The observations were as follows:

(a) Maternal tissue uptake of ^{28}Mg resembled that previously found in nonpregnant adult rabbits with the exception that bone and muscle uptake was slower.

(b) Placental concentration of ^{28}Mg rapidly rose above the maternal plasma level.

(c) By 26 hours the specific activity of magnesium in all the studied fetal tissues reach a fairly constant value.

(d) The turnover of magnesium in fetal tissues *in utero*, particularly in bone and muscle, is considerably more rapid than in the respective maternal tissues.

C. Dogs

1. Smith *et al.* (712) examined the distribution of magnesium in dogs after injection of magnesium sulfate. Adult female dogs (number not given) were injected in the femoral or jugular vein with an isotonic magnesium sulfate solution (0.154 M at a rate of about 10 cc per min.). All urine and feces were collected for the next 24 hours. The concentration of magnesium in the serum preceding and following injection was compared. The retention of any magnesium in the body was determined from these data and the amount of magnesium still remaining in the extracellular fluid calculated. The authors postulated that if some of the retained magnesium could not be accounted for in the extracellular fluid, it would have to have been deposited elsewhere.

It was observed that:

(a) In the first 3 or 4 hours after injection the volumes of distribution of both magnesium and sulfate ions were nearly identical. This indicates that both ions are distributed throughout the same portion of the extracellular fluid (20 to 25% of the body weight). Therefore magnesium distribution resembles that of sodium rather than potassium.

(b) All the injected magnesium could not be recovered in the urine and stools within 24 hours. In fact the authors did not believe there was any evidence that any of the injected magnesium was excreted via the feces. Therefore they concluded that sometime between the 4th and 24th hour after injection a variable proportion of the injected magnesium over and above the amount which can be accounted for in urine and stools, may leave the extracellular fluid and be segregated in an unknown form in undetermined places in the body.

2. Barbour and Winter (038) investigated both the absorbability of magnesium administered as soluble organic and inorganic salts (gluconate, lactate and oxide) and the question of magnesium and calcium storage.

In seventeen feeding experiments with 5 dogs (age and sex not given) a uniform increase of about 1 mg% in serum magnesium was observed when doses of 200 mg/kg BW of oxide, lactate or gluconate were given. Smaller doses were less effective (see Table 16). Since the calcium content of the serum was reduced in only one case, the authors considered this an indication that a single feeding of a large amount of magnesium does not displace calcium from the blood. Another observation suggested by these experiments (see Biochemical Table 16) was that gram for gram the three compounds tested were practically equivalent in their capacity to increase serum magnesium. This is despite the fact that the relative amount of magnesium in these compounds; gluconate, lactate and oxide, is about 1:2:12.

As a result of these experiments as well as other experiments performed in their laboratory by co-workers and quoted in this paper, the authors concluded that:

Table 16 Maximum changes in Serum Ca and Mg After Oral Administration of MgO, Mg gluconate or Mg lactate (038)

Amount of dose (mg/kg)	Oral dose of MgO		Oral dose of Mg gluconate		Oral dose of Mg lactate	
	Ca mg%	Mg mg%	Ca mg%	Mg mg%	Ca mg%	Mg mg%
50			10.9-12.0	4.0-4.0		
			10.6-10.8	4.0-4.0		
			10.1-10.8	4.4-4.4		
100	11.0-11.8	3.6-3.7				
	10.3- 9.5	3.8-3.9				
	10.6-10.9	4.0-4.5				
200	10.1-11.0	4.6-5.2	10.7-11.4	4.4-5.5	10.2-12.8	4.2-5.2
	10.8-12.3	4.3-5.4	10.1-11.1	4.2-5.4	11.0-12.5	4.6-5.2
	10.8-11.4	4.8-6.1*	9.1-10.4	4.4-5.0	10.2-10.8	4.1-5.5
			10.6-10.5*	3.8-4.0*	10.4-10.6*	3.7-4.0*

*Two hours only.

(a) The absorptive capacity for magnesium cannot be regarded as constant. Ordinarily harmless amounts of substances can for example be absorbed from a diseased or irritated intestine with unexpected results.

(b) The form in which magnesium is ingested can determine both its rate and total amount of absorption.

(c) Both soluble organic and inorganic magnesium salts markedly raise the level of serum magnesium. When large amounts of orally administered organic magnesium salts are administered, from one- to two-thirds may be readily absorbed.

A series of feeding experiments with magnesium gluconate and lactate in which the dietary levels of magnesium calcium and phosphorus were varied (adequate to low) was also carried out. From these the authors concluded that:

(a) When the phosphate intake was adequate, prolonged administration of large amounts of soluble organic magnesium salts was not accompanied by loss of serum calcium.

(b) When the phosphate intake exceeded 4 mmoles/kg BW/day, additional calcium was retained. (See original paper for tables.)

D. Humans

1. Wacker and Parisi (309) summarized some of the research findings on magnesium absorption. Magnesium is the fourth most abundant cation in the body (21-28 g in an adult) and the second most plentiful intracellularly. Bone contains about half the total body magnesium. The remainder is almost equally distributed between muscle and the nonmuscular soft tissues. Liver and striated muscle have the highest concentration (15 to 20 mEq/kg) of the non-osseous tissues. The concentration of magnesium in erythrocytes varies from 4.4-6.0 mEq/liter.

Calcium influences the amount of magnesium absorbed from the dietary intake (ca. 20-25 mEq/day) apparently by competition for a common absorptive pathway. About one-third of the daily ingested magnesium is absorbed.

It has been shown from animal studies that magnesium is absorbed mainly in the proximal part of the small intestine. In man the time course of appearance of orally administered labeled magnesium (^{28}Mg) in the plasma is consistent with this interpretation. Peak levels of the isotope are measured between two and eight hours after the dose. However, after rectal enemas, colonic absorption can occur as evidenced by the development of hypermagnesemia (see Biological Section II B).

2. Silver et al. (701) examined magnesium turnover in man with ^{28}Mg. The authors noted that both the use of this isotope and interpretation of results obtained with it should be viewed with caution because of its relatively low specific activity.

Ten adults (white, 3 males and 7 females) ranging in age from 38 to 72, were infused intravenously or orally administered a specially prepared ^{28}Mg solution (pH 7.4 in HCl) in amounts ranging from 20 to 104 μc (total volumes ranged from 25 to 100 ml). Each patient was on a consistent diet for 5 days before and 3 days after administration of the isotope. The magnesium dietary intake was fixed at about 50-60 mEq/day for each patient.

The chief observations following intravenous administration were that the isotope appeared in the urine rapidly but only insignificant amounts appeared in the stool. The authors interpreted this as indicating that most of the magnesium in the stools is of exogenous origin.

At 40-60 hours only about 10-25% of the body's total magnesium had equilibrated with the isotope and at 90 hours about one-third had. Thus indicating that equilibration of the isotope with the magnesium in the body is slow.

The authors concluded that their studies tended to confirm earlier data from magnesium turnover studies with nonisotopic techniques which showed:

(a) There are several relatively small rapidly equilibrating compartments. In man there are at least three compartments in the magnesium body pool which turnover with half-times of 1, 3 and 14 to 35 hours respectively.

(b) There is one or more compartments in which turnover is very slow. In man probably 25-50% of the magnesium has a turnover rate of less than 2 percent per day.

(c) Studies with ^{28}Mg in man suggest that gastrointestinal absorption of Mg^{2+} is very limited.

However, the authors pointed out that these turnover values must be interpreted with caution because of possible effects from the significant amounts of dietary magnesium in addition to the effect of that administered with the low specific activity ^{28}Mg.

3. Hirschfelder (323) studied the effects in humans of taking a single ordinary purgative dose of epsom salts. First they administered this does of magnesium sulfate (within 24 hours after ingestion) to 7 normal men. They found that within 24 hours, 40-44% of the ingested dose appeared in the urine. However, despite the large quantity of absorbed magnesium the concentration of magnesium in their blood plasma remained relatively constant.

However, when a similar oral dose of magnesium sulfate was given to a series of patients (13) with renal disease, a tremendous rise in plasma magnesium occurred within 4-6 hours (from a normal 2 mg/100 cc to as high as 9-11 mg/100 cc) or to about two thirds of the concentration at which coma sits in. The authors concluded that when magnesium sulfate is administered by mouth to patients with renal disease it is absorbed continuously from the intestine. Therefore dangerously high magnesium levels are reached in the blood.

4. Stevens and Wolff (735) attempted to correlate the serum level of magnesium owing to rectal absorption with the clinical state of the subject.

Six volunteer male subjects were administered hypertonic solutions of magnesium sulfate by rectum (maximum of 300 cc). Four of the subjects received a one-half saturated solution and two a saturated solution.

From the results of their experiment as well as reports in the literature, the authors stated that it was obvious magnesium sulfate is absorbed from the gastrointestinal tract including the rectum in sufficient amounts to raise the blood level significantly (see individual curves in original paper) and to produce depressant cerebral effects. They additionally noted that even normally functioning kidneys may not remove the salt from the blood stream fast enough to prevent the accumulation of amounts toxic to the central nervous system.

Normal serum magnesium appeared to be in the range of 1.5-2.5 mg/100 cc.

Relation of serum magnesium level and effects noted

Serum Magnesium Level (mg/100 cc)	Nature of effect
4	sensations of warm skin and dry mouth
7	drowsiness noted
12	marked drowsiness
12-16	diaphragmatic respirations
20.1	respiratory arrest

In another experiment, three subjects were each given a saturated solution of magnesium sulfate orally (40 cc, 25 g). All showed some increase in their serum magnesium (3.6, 4.0 and 4.3 g/100 cc respectively).

Total amount of administered magnesium sulfate and serum magnesium levels

Magnesium sulfate administered (g)	Serum magnesium level (g/100 cc)
25 (orally)	4 (average)
94 (per rectum)	7.0
126 (per rectum)	9.0
158 (per rectum)	16.2
189 (per rectum)	20.1 (post mortem)

The authors concluded that magnesium sulfate can be absorbed from the intact bowel in large amounts and may cause dangerous depression of the central nervous system even when renal function is normal.

5. Graham et al. (271) used ^{28}Mg to obtain further information on the effect of variation of dietary intake on magnesium absorption and excretion in humans.

Ten subjects (8 male and 2 female, 25-64 years old) were fed a control diet (20 mEq Mg/day) and 5 ml of ^{28}Mg (as $MgCl_2$, specific activity 5 uc/mEq) taken with breakfast. Three other subjects (1 female and 2 males) were fed abnormal diets; low Mg, 1.9 mEq/day and high Mg, 47 mEq/day.

Absorption of the ^{28}Mg was studied by both fecal collection and analysis of the plasma radioactivity.

The observations were:

(a) Within an hour following ingestion, absorption began, reached a steady level at 2 to 3 hours and continued relatively constantly for the next 4-6 hours. By this time 80% of the total absorption had taken place.

(b) After 12 hours there was very little absorption.

(c) 44.3% of the ingested radioisotope was absorbed with the average diet (20 mEq Mg/day).

(d) 75.8% was absorbed on the low magnesium diet (1.9 mEq/day).

(e) 23.7% was absorbed on the high magnesium diet (47 mEq/day).

The authors concluded that:

(a) There is fairly uniform absorption throughout the small intestine.

(b) There is little or no absorption from the large bowel.

(c) Absorption is affected by the amount presented to the intestinal mucosa but the relationship is not linear.

(d) Absorption is apparently unaffected by depletion or repletion of body magnesium.

III. Metabolism and Excretion

A. Rats

1. Clark (142) studied the effects of orally administered supplemental, non-toxic amounts of magnesium chloride on calcium and phosphate metabolism in adult rats. A basic diet containing the same amount of magnesium (0.05%) was fed to 144 adult male Holtzman rats (275-295 g, divided into 12 groups). The diet of each group contained different percentages of calcium and phosphorus (40% of diet) as mixtures of various salts of these elements (see Table 17) after 5 days of habituation to the diets, each of the groups was subdivided into two subgroups of 6 animals. A 10% (w/v) glucose solution was added to the drinking water of all the rats. One group of each of the subgroups was also given magnesium chloride in the drinking water (final concentration 2% w/v).

A balance study was carried out for 12 days. During this time the control animals consumed 50 mg of magnesium (as $MgCl_2$) while the experimental animals consumed 800-1000 mg. As a result of the data developed from analysis of both the food and fluid consumption and the urine and feces the following conclusions were made:

(a) Provided the diets contained at least 0.4% calcium, supplemental magnesium ions decreased fecal calcium significantly.

(b) Supplemental magnesium ions significantly increased urinary calcium in all groups.

Table 17. Dietary calcium-to-phosphorus ratios (142)

Percent Dietary Phosphorus			Percent Dietary Calcium			
			0.2	0.4	0.8	1.6
	0.8	KH_2PO_4	14.5	14.5	8.0	8.0
		Na_2HPO_4			3.7	3.7
		$NaH_2PO_4 \cdot H_2O$	7.1	7.1		
		$CaH(PO_4)_2 \cdot H_2O$	12.6	12.6	23.5	
		$Ca_3(PO_4)_2$				28.9
		$CaCO_3$		5.0	10.7	12.0
	0.4	K_2HPO_4			8.0	8.0
		KH_2PO_4	10.5	10.5		
		$KHCO_3$	1.5	1.5		
		Na_2HPO_4			3.7	3.7
		$NaH_2PO_4 \cdot H_2O$	7.2	7.2		
		$CaH(PO_4)_2 \cdot H_2O$			7.3	7.2
		$CaCO_3$	5.0	10.0	17.1	37.3
	0.2	K_2HPO_4	5.2	5.2	6.7	6.7
		$KHCO_3$	5.4	5.4		
		KCl			1.1	
		$NaH_2PO_4 \cdot H_2O$	3.6	3.6		
		$NaHCO_3$	2.2	2.2		
		$NaHPO_4$			3.7	3.7
		$CaCO_3$	5.0	10.0	20.0	40.0

(c) Supplemental magnesium ions affected fecal phosphorus as follows: (a) no effect when dietary phosphorus was low (0.2%), (b) caused a decrease when dietary phosphorus was 0.4%, (c) caused an increase when dietary phosphorus was high (0.8%) and calcium low (0.2%).

Magnesium ions were found to lower serum calcium. While at low dietary levels of phosphorus (0.2%) the magnesium-phosphorus interaction had the effect of lowering serum calcium.

The conclusions drawn from this study were:

(a) Dietary magnesium ions markedly influenced the absorption and excretion of calcium and phosphorus. These effects of magnesium were also dependent on the dietary levels of calcium and phosphorus, i.e., calcium absorption was increased only when the diet contained 0.4% or more calcium.

(b) The increase in intestinal absorption of calcium was reflected in elevated urinary calcium. The authors noted that other studies have shown that alkaline magnesium salts such as the oxide or hydroxide are as effective as the acidic chloride in producing hypercalciuria.

(c) The improvement in phosphorus balance as a result of supplemental dietary magnesium appears to be a consequence of renal conservation of phosphorus.

Two possible consequences for human beings were pointed out by the authors:

(a) Since divalent magnesium has also been shown to increase calcium balance and absorption in man, it might be of value in the treatment or prevention of those metabolic bone diseases which result from inadequate calcium absorption from the intestines.

(b) A partial explanation for the reduction of renal calculus formation in man following the oral administration of magnesium ions may be the observation in this study that supplementary dietary magnesium ions resulted in a fall in urinary phosphorus. (See also this section D, 6 and Biochemical Section V, C, 1.)

2. MacIntyre et al. (464) studied the effect of parathyroid hormone on the urinary excretion of magnesium ions in the rat. Pure bovine parathyroid hormone (2000 units/mg potency) was infused (10 μg) into the tail vein of male parathyroidectomized rats (150 g) along with an infusion solution containing: glucose, 44 g/liter; sodium chloride 19 mEq/liter; calcium lactate, 10 mEq/liter; magnesium chloride, 10 mEq/liter: and sodium heparin, 0.1 g/liter. A marked fall in magnesium excretion was observed. An effect was noted on magnesium excretion when as little as 1 μg of the hormone was infused. The authors claimed that these experiments conclusively show that parathyroid hormone causes marked conservation of urine magnesium. They attribute this to a direct renal effect owing to a change in the tubular handling of the magnesium cation.

B. Dogs

1. Speranskaja-Stepanowa (725) studied the influence of magnesium on renal activity in dogs. Dogs with chronic fistulas of the ureter (3, sex and age not given) were injected with crystalline $MgSO_4$ in 20% and 50% solutions. The doses ranged between 0.08 and 0.4 g/kg BW. Most of the doses administered were a 50% solution of 0.08 g/kg BW. At this dosage no effect on urinary excretion was observed. Doses higher than 0.4 g/kg caused a distinct reduction in the quantity of urine.

2. Haury and Cantarow (304) studied the effect of magnesium sulfate on calcium, magnesium and inorganic phosphorus in the serum, and peritoneal

fluid of dogs. Of the four adult dogs used in the experiment, three were injected intramuscularly and one subcutaneously with 50 cc of a 25% magnesium sulfate solution.

The effects observed on the serum and peritoneal fluid were:

(a) A fall in serum Ca but a relatively insignificant change in peritoneal fluid Ca concentration.

(b) A fall in serum inorganic P and a simultaneous fall in peritoneal fluid P.

(c) Tetany occurred in 3 dogs in which the serum Ca concentration fell below 8 mg%. It was relieved by intravenous injection of a calcium compound. The authors noted that an explanation for the hypocalcemic tetany was not apparent particularly in the presence of marked Mg concentration in both the serum and peritoneal fluids (also presumably, in tissue fluids).

3. Massry et al. (486) evaluated the characteristics of the renal handling of magnesium in dogs. A variety of test procedures were used (32 experiments) with 30 female mongrel dogs (12-27 kg) in which magnesium chloride or magnesium sulfate added to 15% dextrose in water was infused to deliver 1.0-3.0 μg Mg/min./kg BW over 3-6 hours as follows:

(a) Magnesium chloride or magnesium sulfate alone (8 dogs, 10 experiments).

(b) Simultaneous infusion of 0.9% NaCl and $MgCl_2$ (3 anesthetized dogs).

(c) Simultaneous infusion of 0.9% NaCl, $CaCl_2$ and $MgCl_2$ (3 anesthetized dogs).

(d) Simultaneous infusion of 0.9% NaCl and $MgCl_2$ in dogs pretreated with deoxycorticosterone acetate (DCA) (2 anesthetized dogs).

(e) Infusion of $MgCl_2$ in dogs pretreated with DCA (5 anesthetized dogs).

(f) Simultaneous infusion of $CaCl_2$ and $MgCl_2$ (5 anesthetized dogs).

(g) Parathyroid extract administered during $MgCl_2$ infusion (4 anesthetized dogs). (See original paper for details of these experiments.)

The results of these experiments indicated the following:

(a) Magnesium excretion is determined by filtration and reabsorption alone without evidence for tubular secretion.

(b) There is a maximal tubular reabsorptive capacity (Tm) for magnesium of approximately 140 μg/min/kg BW.

(c) Extracellular volume expansion produced by saline infusion, calcium infusion and chronic treatment with DCA are each associated with a decrease in Tm for magnesium.

(d) Parathyroid hormone may directly enhance magnesium reabsorption.

C. Pigs

Miller et al. (508) determined both the effects of dietary magnesium on the utilization of calcium, phosphorus and magnesium by the baby pig and the magnesium requirement of the baby pig. When the baby pigs were 4-6 weeks of age, 29 mineral balance studies were made.

Calcium, phosphorus and magnesium balance studies were conducted with pigs receiving 75, 225, 325, 425 or 825 ppm magnesium in their diet. Dietary levels of Ca and P were 0.8 and 0.6% respectively. Distilled water was the sole source of drinking water. Data obtained from this study indicated that increasing the dietary level of magnesium did not significantly affect daily Ca or P retention but increased daily Mg retention. The balance data indicated that the dietary Mg requirement is no higher than 325 ppm.

D. Humans

1. Chesley and Tepper (136) studied some of the effects of magnesium acetate and sulfate on the renal excretion of electrolytes in women. The subjects of the study were 34 women, 5 nonpregnant and 29 who were pregnant with a wide range of inulin clearances. Magnesium acetate (100 ml of 10% solution; 93.2 mEq Mg) or magnesium sulfate (16.2 mEq Mg in 10% solution) were injected in various ways (see original paper for details). The results of these experiments were:

(a) The magnesium clearance increased as a roughly linear function of the serum magnesium concentration. The acetate or sulfate anion had little or no apparent effect on magnesium excretion.

(b) The corrected ratio of magnesium clearance (corrected for protein binding of serum magnesium) to inulin clearance approached 1 at the highest levels of magnesium studied. This ratio was roughly a linear function of the serum "filterable" magnesium.

(c) The injection of magnesium salts was followed by an increased urinary calcium excretion. There was a direct relation between

the increased calcium excretion and the serum magnesium concentration.

(d) As the serum magnesium concentration rose, sodium and chloride excretion was stimulated. The excretion of sodium and chloride dropped back to or below control levels when the serum magnesium became constant or fell.

(e) The effect of magnesium on potassium excretion varied from subject to subject.

2. Aikawa et al. (005) used ^{28}Mg to explore the kinetics of magnesium distribution in normal subjects and in patients with various diseases. A total of 27 studies were performed on 9 normal subjects and 16 patients with various diseases. The ^{28}Mg was administered intravenously in 12-30 mEq of stable magnesium in 250-350 ml of 5% aqueous dextrose. Urine samples and blood samples from the brachial vein were collected at intervals (most over 24 hours; a few blood samples over 37 hours and urine samples over 67 hours).

The observations in normal subjects were:

(a) Plasma disappearance was rapid within the first several hours.

(b) Within 24 hours, a mean of 19.8% of the injected radioactivity was accounted for in the urine.

(c) Within 18 hours equilibration of ^{28}Mg in the bile occurred but fecal excretion was negligible.

(d) The specific activities of plasma and urine stabilized by the 18th hour and thereafter showed only a gradual decrease.

(e) Exchangeable magnesium contents ranged between 2.6 and 5.3 mEq/kg BW (less than 16% of the estimated total magnesium content of the body).

(f) ^{28}Mg exchanged very slowly with the stable ion in bone, muscle and erythrocytes.

There were no striking differences between these results with normal patients and those obtained from patients with diabetes mellitus and hepatic diseases.

The authors summarized the knowledge concerning magnesium metabolism in human beings at the time they wrote their paper (1960) as follows:

(a) The fraction of magnesium absorbed when it is introduced into the body by the gastrointestinal route is considerably smaller than had been thought.

(b) Magnesium metabolism appears to be related to the metabolism of phosphorus and glucose in which the liver appears to play a role.

(c) Whether or not there is a specific hormonal regulatory mechanism is not known.

(d) The labile pool of magnesium in the body, a small fraction of the total body content of magnesium, is confined primarily to the soft tissues.

(e) Magnesium exchange in bone, muscle, and erythrocytes is extremely slow.

(f) Parenterally administered magnesium is excreted very slowly by the kidneys. The over-all balance however, is maintained by a compensatory rapid excretion of endogenous magnesium.

3. Consolazio et al. (151) studied magnesium excretion in sweat. The study was conducted on 3 healthy young men for a total of 32 days.

The men were exposed on a balance study for 16 days to a 37°C temperature for 7.5 hours. It was found that from 10.1 to 15.4 percent of the total output of magnesium was via sweat. The magnesium loss amounted to about 17 mg per day. Acclimatization did not occur as it did in the cases of sodium and potassium. The authors concluded that under extreme conditions sweat could account for about 25% of the magnesium lost daily. When intakes are low this could be important.

4. Ozsoylu (565) included a discussion of magnesium metabolism in his paper. Briefly he noted:

(a) As an intracellular cation known to be an activator in several pathways of carbohydrate metabolism, magnesium is second in abundance only to potassium.

(b) Figure shows some of the pathways in which the magnesium ion has some role such as Embden-Meyerhof, glucose storage and pentose shunt.

(c) The magnesium ion is also required in the citric acid cycle:

$$\text{Succinyl CoA} \xrightarrow{Mg^{++}} \text{succinate}$$

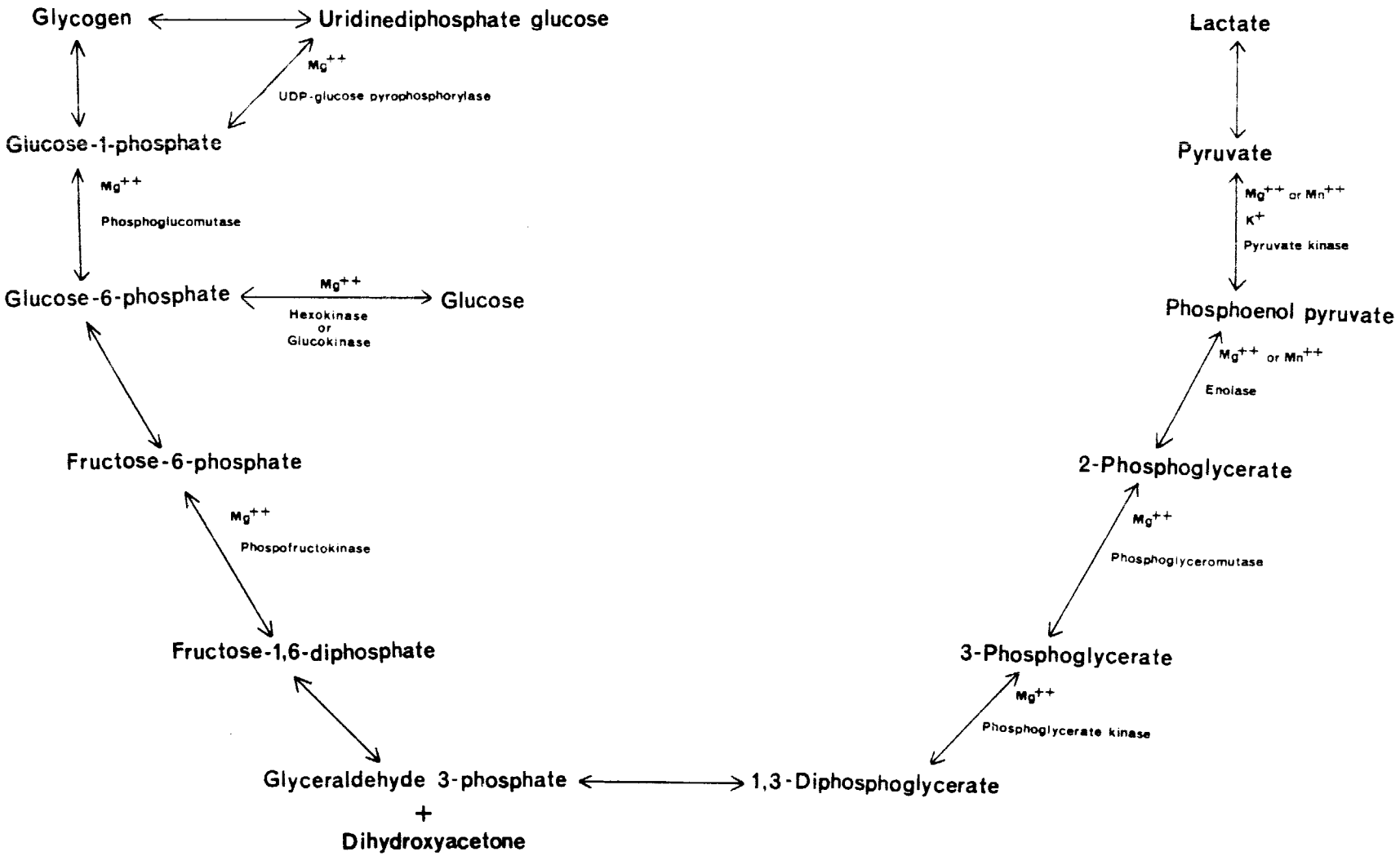
(d) The magnesium ion is known to be necessary for the activation of ATP requiring enzymes as well as phosphoralase, phosphatase, enolase, peptidase and decarboxylase.

(e) An absolute requirement for this ion, has however, not been proven.

5. Jones et al. (364) investigated the relationship between the functional status of the thyroid gland and magnesium metabolism. Hyperthyroid and hypothyroid patients were studied by determinations of serum and erythrocyte magnesiums, exchangeable magnesiums, and by complete

Figure 1.

Enzymatic Systems Requiring Mg



balance studies during therapy with propylthiouracil and triiodothyronine, respectively. Eight hyperthyroid and eight hypothyroid patients were used in this study which was carried out with ^{28}Mg. At zero time 50-150 uc of ^{28}Mg was injected intravenously over a 2-3 minute period. The results of serum urine and stool analyses showed that:

(a) Plasma magnesium values tended to be lowered in hyperthyroidism and elevated in hypothyroidism.

(b) Erythrocyte magnesium levels were significantly reduced in only one hyperthyroid patient before therapy and varied predictably after therapy.

(c) Hyperthyroid patients were found to have decreased plasma magnesium, increased urinary excretion of ^{24}Mg and ^{28}Mg and normal total and cellular exchangeable magnesium before therapy.

(d) Hypothyroid patients had elevated plasma magnesium, decreased urinary ^{24}Mg and ^{28}Mg excretion, increased fecal magnesium excretion, and strikingly reduced total and cellular exchangeable magnesium before therapy.

(e) Erythrocyte magnesium values were normal in both groups.

The authors concluded that the data suggest that a defect in magnesium transport occurs in thyroid hormone deficiency states.

6. Drake (200) followed the effects of orally administered magnesium oxide (MgO) on urinary Ca and P excretion and attempted to ascertain whether MgO exerts a "protective effect" in calcium oxalate stone formation. Forty healthy young men on a submarine patrol were the experimental subjects. Urinary samples were taken at the beginning and end of the patrol. During the intervening period twenty-one of the men ingested

310 mg MgO daily, while nineteen served as controls. The test group was found to excrete significantly more calcium than the controls. The author postulated that magnesium oxide exerts its protective influence on renal stone formation by means of a mechanism whereby the rate of calcium excretion is altered. He also noted that this finding does not agree with the "decreased urinary excretion" theory for the protective influence, but rather lends some support to the "magnesium-oxalate balance" theory. (See also Biochemical Section V, C, 1 and this section A, 1.)

IV. Enzymes and Other Biochemical Parameters

A. In Vitro

1. MacIntyre (463) summarized the *in vitro* actions of magnesium ion in a review. He noted however, that so many enzymes are likely to be affected that predicting *in vivo* effects of magnesium deficiency from the *in vitro* actions of the ion is quite impossible.

The enzyme systems for which magnesium is important are:

(a) Magnesium activates the alkaline phosphatases, pyrophosphatases and prostatic acid phosphatases.

(b) Magnesium activates all enzymes transferring phosphate from ATP or to ADP. The author noted that the reactions involving ATP and ADP are so widespread that magnesium must influence all life processes.

Examples of the phosphate-transferring enzymes activated by magnesium are: hexokinase, fructokinase, creatine transphosphorylase, phosphopyruvic transphosphorylase, diphosphopyridine nucleotide phophorylase and phosphoglucomutase.

(c) Magnesium ions are necessary whenever diphosphothiamine is required. In this group are: yeast carboxylase, mammalian heart carboxylase and the pyruvic oxidase system of brain.

(d) Enolase, which catalyses the interconversion of 2-phosphoglyceric and phosphoenol-pyruvic acids, generating an energy-rich phosphate bond in the process, requires high concentration of magnesium.

(e) Some peptidases, such as leucine aminopeptidase, require magnesium.

(f) Many of the reactions of glycolysis are activated by magnesium ions.

2. Wacker and Parisi (809) summarized the information on the enzyme systems critical to cellular metabolism for which magnesium has been found to be an activator in vitro. It is involved as follows:

(a) The enzymes that hydrolyze and transfer phosphate groups: e.g. the phosphatases and the enzymes concerned in reactions involving adenosine triphosphate (ATP) are activated by magnesium.

(b) It is a co-factor for oxidative phosphorylation.

(c) The highly ordered organization of DNA, RNA and ribosomes is stabilized by the presence of magnesium.

(d) The aggregation or dissocciation of component particles in ribosomes obtained from Escherichia coli, baker's yeast, pea seedlings, rat liver and rabbit reticulocytes is critically dependent on the ambient magnesium concentration.

The authors noted in this regard that the fundamental importance of magnesium in cellular function is suggested by its analogous role in plant and animal systems and the generality of this phenomenon.

(e) It is also required for the *in vitro* synthesis and degradation of DNA.

(f) The first step in all the amino acid activating systems, in which a specific amino acyl sRNA synthetase forms a complex with its amino acid in the presence of ATP, is known to be magnesium dependent. The second or transfer step in which a specific sRNA accepts its amino acid from the enzyme complex is not ordinarily magnesium dependent but becomes so if the sRNA is denatured in a manner which removes the metals it contains naturally. The two steps in the formation of aminoacyl sRNA are:

I. Activation

$$\text{Enzyme} + \text{Amino Acid} + \text{ATP} \underset{}{\overset{Mg^{++}}{\rightleftharpoons}} \text{Enzyme-Amino-Acyl-Adenylate} + \text{PP}$$

II. Transfer

$$\text{Enzyme-Amino-Acyl-Adenylate} + \text{sRNA} \rightleftharpoons \text{Aminoacyl sRNA} + \text{Enzyme} + \text{AMP}$$

The authors concluded from all of the above observations that since magnesium is the most abundant divalent intracellular cation that it would be a tenable hypothesis to assume analogous *in vivo* function.

3. Lebduska and Cervinka (434) examined whether colloidal magnesium hydroxide causes an increase in phagocytosis *in vitro*. White corpuscles from horse venous blood were used. The experiments were performed in test tubes containing: 2 cc of the separated and washed white cells (largely polynuclear); 2 cc of the solutions being studied (colloidal $Mg(OH)_2$ in four different concentrations as shown on Table 18); and 0.25 cc of a *B. coli* culture in peptone containing broth. One tube without $Mg(OH)_2$ acted as a control. The method used was first to count 300 polynuclear cells at each concentration and then determine the number of bacteria phagocytised. Table 18 shows the results. The numbers indicate the phagocytoses per 100 leucocytes.

Colloidal magnesium hydroxide was shown to be a strong stimulator *in vitro* of phagocytosis.

Table 18. Magnesium Effect on Phagocytosis (434)

Experiment	Substance studied	Concentration of the solution Control	N/10	N/100	N/1000	N/10,000
1	$Mg(OH)_2$	20	30	49	42	57
2	$Mg(OH)_2$	90	46	164	210	176
3	$Mg(OH)_2$	42	29	51	59	66
4	$Mg(OH)_2$	25	26	53	59	59
5	$Mg(OH)_2$	65	--	100	164	140

B. Rats

MacIntyre et al. (464) studied the relation of parathyroid hormone to magnesium homeostasis. The details of this experiment are given in Biochem Section III, A, 2.

An important conclusion drawn by the authors was that parathyroid hormone causes marked conservation of urine magnesium and calcium. They further suggested the hypothesis that the mechanism by which plasma magnesium is maintained in the body is the variation in secretion of parathyroid hormone in response to changes of plasma magnesium.

C. Guinea Pigs

Frommel et al. (239) studied the changes in serum cholinesterase when magnesium chloride was injected in guinea pigs. Their experiment is summarized in Table 19.

The authors pointed out that the liver is the most important organ in the regulation of serum cholinesterase. Furthermore, they noted that in producing toxicity in the guinea pig, by injecting inorganic ions, the various functions of the glandular parenchyme have been severely disturbed resulting in anatomical problems.

As can be seen from the tabulated results, magnesium chloride activates cholinesterase. The authors found this effect of interest in contributing to the understanding of the pharmacodynamics of magnesium chloride as an ionic mineral medicine.

D. Dogs

1. LaBarre and Vesselovsky (421) continued their research on the action of hypnotics on the digestive glands by determining the influence of magnesium sulfate on the functioning of the exocrine portion of the pancreas.

Table 19. Magnesium Effect On Serum Cholinesterase (239)

Ions	Guinea pig	Weight	*Dose received in g/kg	CHE before	CHE after	Result in %	Average
Magnesium chloride	m	410	4 x 1,0	2,40	3,60	+33%	
	m	430	5 x 1 g	2,05	2,75	+34%	+34.5%
	m	570	2 x 1 g	1,40	1,90	+36%	

*The injected dose was calculated according to Aberhalden (Handbook of Biological Work Methods 1, 7) to obtain the chronic toxicity for the ion studied.

The experiments were carried out on dogs kept constantly secreting by two means: (1) by continuous intrasaphenous injection of a diluted secretin solution or (2) by administering a test dose of secretin both before and after injecting the magnesium sulfate.

In the first series, there were four experiments in which secretin was injected in chloralosed dogs with a pancreatic fistula (previously ligatured to prevent the passage of chyme into the duodenal region). Once secretion was stabilized 100-150 mg% magnesium sulfate in 20 cc distilled water was injected intrasaphenously.

The results showed the following:

(a) Weak doses of magnesium sulfate (100 mg/kg) resulted in a slight but brief stimulation of pancreatic secretion.

(b) The reduction of pancreatic secretion became more marked at elevated doses.

(c) There was very little variation of lipase content compared to the amount secreted per cc. Generally speaking, the enzyme content of the pancreatic juice varied in proportion to the quantity of juice secreted in a given time.

In a second series of experiments there were two observations:

(a) When the secretin (10 mg/kg) was injected twice at 60 minute intervals in chloralosed dogs with a pancreatic fistula, the secretory responses in a given dog were constant.

(b) After an intrasaphenous dose of secretin when magnesium sulfate was injected, weak doses (100 mg) did not particularly modify the pancreatic secretory response. When the magnesium

sulfate doses reached 150 mg there was a definite decrease of the pancreatic secretory response in two cases.

In this experimental series, the lipase content of the collected juice was reduced.

A third series of experiments were designed to gain understanding of the cause of the decrease in pancreatic secretory ability observed after magnesium sulfate administration. Secretin was administered intravenously to vagotomised dogs with a medullary section at the level of the third cervical vertebra. When constant pancreatic secretion was established, magnesium sulfate was administered in doses of 100-150 mg/kg. The results of four experiments indicated that when the pancreas is not under the control of the central nervous system, magnesium sulfate does not reduce pancreatic secretion.

The authors concluded from their experiments that:

(a) Magnesium sulfate has a depressive effect on gastric and pancreatic secretions.

(b) The inhibition of postinsulin gastric hypersecretion can be attributed to a postmagnesium paralysis of the thalamic centers.

(c) The observed blocking of the pancreatic exocrine function apparently is dependent on the integrity of the encephalic centers.

(d) In those persons in whom pancreatic function is cut-off by administering secretin or who have food in the digestive tract, magnesium sulfate reduces pancreatic juice production and enzyme formation. This action is similar to that of the opiate hypnotics.

2. LaBarre and Kettenmeyer (419) studied whether magnesium sulfate modifies postinsulin hyperadrenalinemia by paralysing the thalamic centers. Lightly chloralosed dogs (number, age, and sex not given) were intravenously injected with 100-150 mg/kg BW of magnesium sulfate. In order to evaluate the adrenaline secretion quantitatively, a fistula was made in the suprarenal vein. Samples of blood were taken and adrenalinemia was evaluated by studying the comparative effect of various blood samples on intestinal motility. (See Figures 1 and 4 in original paper for further details.)

The authors concluded from these experiments, that moderate doses of magnesium sulfate (100-150 mg/kg) inhibit postinsulin hyperadrenalinemia by paralysing the thalamic centers.

V. Drug Interaction

A. Rats

Haggard and Greenberg (292) investigated the potential of magnesium sulfate as an antidote for strychnine poisoning. In the experiment recorded in Figure 2, 23 rats (age, sex and strain not given) were injected peritoneally with strychnine in varying amounts from 0.001 to 0.004 mg/g BW. Magnesium sulfate solution was injected intraperitoneally in varying dosages (unconsciousness was caused at 0.8 mg/g BW and death at 0.9 mg/g BW and above). As can be seen in Figure 2 the antispasmodic, magnesium sulfate, did not either prevent or even diminish strychnine convulsions in rats. Magnesium sulfate is therefore not an antidote for strychnine.

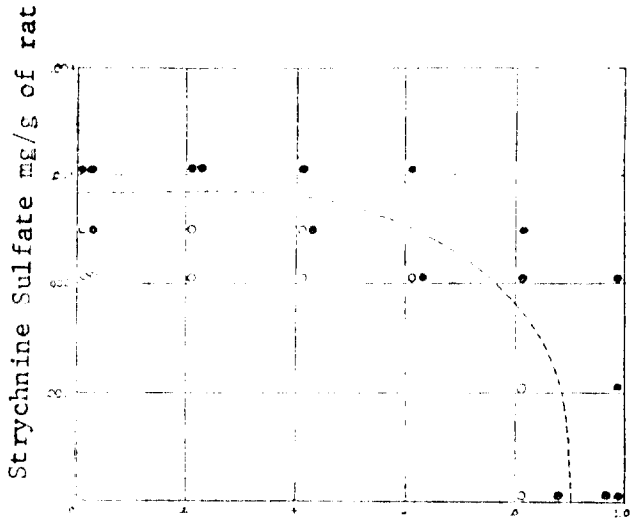


Fig. 2. Magnesium sulfate administered to rats poisoned with strychnine: The rats represented by the black dots died; those represented by circles lived. The dose of strychnine and apomorphine given any one of the rats is indicated by the points on the abscissa and ordinate corresponding to the rat. Magnesium sulfate is not an antidote for strychnine. (292)

B. Rabbits

1. Barbour and Taylor (039) studied the relations obtained between various combinations of sodium barbital and magnesium chloride with respect to toxicity and hypnotic action. Both compounds were administered subcutaneously to normal, healthy adult rabbits. Table 20 shows the toxicity of the two drugs separately and combined.

Table 21 shows the narcotic effects of combining sodium barbital with magnesium chloride as compared to those obtained with sodium barbital alone. From this table it can be seen that it took about one-third longer for the onset of either light or deep narcosis with sodium barbital alone than with a 1:2 combination of sodium barbital and magnesium chloride. Recovery as well as narcosis began much more slowly with the 1:2 combination. Partial recovery required about one-third as long and complete recovery about two-thirds as long as with sodium barbital alone. The phenomenon of accelerated onset and accelerated recovery from barbital narcosis occurred over a wide range of dosage and combination ratios. These different ratios are indicated on Figure 3 .

The authors speculated that the observation that when magnesium chloride is added to sodium barbital recovery is hastened, is probably due largely to the relative rapidity of both absorption and excretion of the smaller magnesium chloride molecule. They further suggested that the "protective antagonism" observed could be accounted for both by the difference in time of peak effect as well as by differences in the anatomical loci of toxicity.

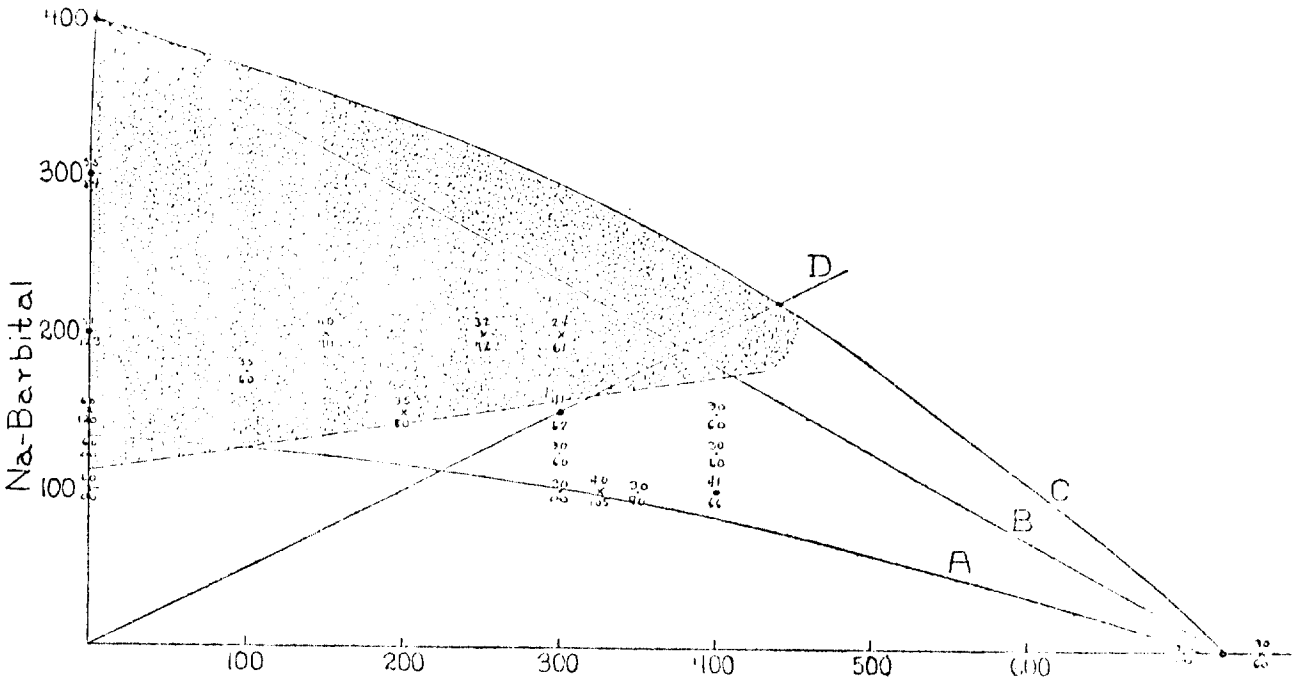
Table 20. Toxicity For Rabbits (039)

Na BARBITAL		$MgCl_2$	SURVIVED	DIED	FATALITY
mgm. per kgm.		*mgm. per kgm.*			*per cent*
350					0 } Roetner (9)
400					100 }
		700	11	1	9
		725	10	6	60
		750	2	3	60
		775	0	2	100
188	+	376	2	0	0
192	+	385			Predicted m.l.d.
196	+	392	2	0	0
212	+	424	4	0	0
220	+	440	2	2	50
230	+	460	1	1	50
240	+	480	3	1	25

Table 21. Na-Barbital Narcosis With and Without $MgCl_2$ (039)

NUMBER OF EXPERIMENTS	AMOUNT INJECTED		M.L.D.	AVERAGE TIME REQUIRED TO PRODUCE			
	Barbital	$MgCl_2$		Light narcosis	Deep narcosis	Partial recovery	Complete recovery
	mgm. per kgm.	*mgm. per kgm.*	*per cent*	*minutes*	*minutes*	*minutes*	*minutes*
12	200	0	50	51 ±2.9	123 ±22.1	>167	>600
10	300	0	75	55 ±2.6	84 ± 3.8	>696	>810
12	150	300	68	41 ±1.9	62 ± 2.0	233†	560†
7	100	400	81*	41 ±4.5	66 ± 2.7	281	475

* Of predicted.
† Eight rabbits only.



Magnesium Chloride

Fig. 3. Rabbit Narcotic and Toxic Dosage of Solium Barbital, Magnesium Chloride and Their Combinations (039)

Ordinates: Sodium barbital milligrams per kilogram; abscissa: magnesium chloride mg/kg. A, threshold dosage for deep narcosis; B. toxicity line (predicted or additive); C, toxicity line based on findings with Na-barbital plus $MgCl_2$ in 1:2 ratio; small dots, results from individual rabbits; crosses, average result from two rabbits; large dots, average results well established by numerous experiments (see tables 20 and 21). Line D represents all sub-lethal doses of a combination of one part sodium barbital to two parts of magnesium chloride. Small figures above points indicate minutes required to produce light narcosis; figures below points, minutes required to produce deep narcosis; (infinity = no deep narcosis). Long dash line, threshold dose for complete recovery within ten hours (shaded area longer persistence of action).

The main conclusions of this study were:

(a) A combined single injection of magnesium chloride and sodium barbital when given to rabbits can both hasten the onset of narcosis and lessen the persistence of the narcotic effect without evidence of significantly increasing toxicity.

(b) When two parts of magnesium chloride are combined with one part sodium barbital, the minimum lethal dose is considerably higher than would be predicted on the basis of simple summation of effects.

2. Barbour and Winter (037) investigated the antipyretic action of magnesium chloride alone and combined with amidopyrine (pyramidon) in rabbits.

Seventeen healthy adult rabbits were made feverish (1.5°F increase in 2 hours) by subcutaneously injecting 1 cc/kg BW of hay-infusion. Doses of magnesium chloride in 5% solution ranging from 150-300 mg/kg BW were administered to 12 of the rabbits with 5 as controls. It was found that definite magnesium antipyresis was produced with a dose of about 200 mg/kg BW and above of the chloride. The authors noted that this was well below the minimum lethal dose of 700 mg/kg BW previously determined by them.

In another series of experiments magnesium chloride was administered in varying combinations with amidopyrine to 18 rabbits. (See original paper for details.)

The authors found that the combination of a weak amidopyrine dose (100 mg) with a weak magnesium chloride dose (150 mg) produces a profound drop in temperature. The maximum decrease in 9 rabbits was 3.9°F. This

was a greater effect than was produced by separate doses of either 200 mg/kg amidopyrine or 300 mg/kg magnesium chloride.

The authors concluded from their study that combinations of magnesium chloride with amidopyrine showed both higher therapeutic efficiency and lower toxicity than amidopyrine alone.

3. Winter and Barbour (831) studied the effect of magnesium chloride on the antipyretic action of sodium salicylate. When 150 mg/kg BW magnesium chloride (ineffective alone) was subcutaneously administered (5% solution) combined with 50 mg/kg BW sodium salicylate (5% solution) to 13 fevered rabbits and combined with 100 mg/kg BW sodium salicylate to 8 fevered rabbits, rapid reduction in temperature was noted. Control fevered rabbits (17) were used to judge the temperature difference.

It was observed that:

(a) The combination of 50 mg/kg BW sodium salicylate with 150 mg/kg BW magnesium chloride was more effective than 100 mg/kg BW sodium salicylate administered alone.

(b) The slow, extended temperature fall produced by salicylate was modified to a rapid but briefer effect with the addition of magnesium chloride.

The authors concluded that magnesium salts potentiate the early effects of antipyretic drugs. This synergistic effect takes place even when intense or prolonged action tends to be inhibited.

C. Dogs

1. Winter and Barbour (831) investigated the effect on the antipyretic action of aspirin when combined with magnesium chloride or magnesium oxide.

They found that the combination of 150 mg/kg BW magnesium chloride and 100 mg/kg BW aspirin reduced the fever of 4 dogs as effectively as 150 mg/kg BW aspirin alone.

The combination of magnesium oxide with aspirin (100 mg/kg BW aspirin plus 100 mg/kg BW MgO with 5 fevered dogs) was not as effective as an antipyretic as 150 mg/kg BW aspirin alone. A study of magnesium oxide alone showed that it had a weaker antipyretic action than magnesium chloride (see original paper for curves). The authors attributed this to slower absorption of the oxide as compared to the chloride.

2. Winter et al. (832) studied whether the antipyretic action of phenacetin in dogs would be increased by simultaneous oral administration of magnesium oxide. The dogs were made feverish by injection with fresh hay infusion.

When MgO in doses from 75-300 mg/kg BW was orally administered to 15 dogs, 6 of them showed an antipyretic effect which was not dose related. When 200 mg/kg BW phenacetin was orally administered combined with 100 mg/kg BW MgO to 11 fevered dogs, the antipyretic effect was as great as 300 mg/kg BW phenacetin given alone.

The authors concluded that the antipyretic effect of orally administered phenacetin was enhanced by the addition of MgO. They considered their results as strongly indicative of synergism since large doses of MgO alone were not antipyretic.

D. Humans

Gershoff and Prien (253) investigated the effects of the daily administration of magnesium oxide (MgO) and vitamin B_6 (pyridoxine) on patients with histories of recurring calcium oxalate urolithiasis (see also Biochemical Section III, D, 6).

Male and female adult patients with a two-year history of forming two or more calcium oxalate renal stones yearly, were used in this study. The subjects were asked to take two 100 mg MgO tablets and one 10 mg pyridoxine tablet daily. Thirty-six patients remained in the program for 5 years.

After one year urine and serum constituents showed the following effects:

(1) The urines of 45 of 51 patients showed an increased ability to hold oxalate in solution.

(2) Urinary calcium and citric acid were raised.

(3) Urinary xanthurenic acid was lowered.

(4) Urinary magnesium, oxalic acid, phosphate-P or pyrophosphate-P showed no significant changes in values.

(5) Serum magnesium was lowered.

(6) Serum beta-lipoproteins decreased in 25 of the 51 subjects.

After 5 years, 30 out of 36 patients maintained on the program showed either no recurrence or decreased recurrence of stone formation.

The authors postulated that a possible explanation for the protective action of MgO and vitamin B_6 administration in preventing renal stone formation might involve an effect on the solvent characteristics of urine. They also pointed out that since there is no reason to believe there is a common etiology in all cases of calcium oxalate urolithiasis, a variation in individual response to prophylactic treatment would be expected.

VI. Consumer Exposure

As discussed in the Chemical Section VIII, magnesium is a required co-factor for many enzymes, and is also required by plants in the course of photosynthesis. By virtue of these facts, magnesium is found at some level in all vegetables, fruits, meats and seafoods. Table 22 from Schroeder et al. (677) gives magnesium values for a wide variety of foods on both a weight and a caloric basis. (Analyzed by atomic absorption spectrophotometry.)

In addition to exposure to magnesium as a natural constituent of fruits, vegetables, dairy products, meats, and seafood, magnesium may also be encountered as a result of the usage of various magnesium compounds in food processing. Of these compounds, magnesium carbonate is produced in the greatest quantity, with almost 500,000 pounds being produced in the United States in 1970, as shown by an NAS/NRC survey (Table 23).

Magnesium carbonate, in addition to its antacid and laxative uses, is employed in the processing of a variety of foods and may serve as an anti-caking and drying agent, a carrier, a disintegrating and dispersing agent, a color fixative, a color retention agent, and as an adjunct. Magnesium carbonate, in conjunction with sodium phosphate and an emulsifier, has been used in attempts to reduce the amount of chlorophyll degradation in green plants (038). Magnesium carbonate has reportedly been used as an adulterant in pepper improving the appearance of the pepper (206). Also, not too infrequently, it has been added to baking powders for the purpose of adding deceptive bulk.

Table 22. Magnesium in Foods (wet weight) (677)

	μg/g		mg/100 kcal
Fish and sea food			
Blue Gill, raw, frozen	375		31.7
Cod, raw, frozen (3)	249	(207–285)	30.4
Haddock, raw, frozen (2)	484	(436–532)	68.2
Haddock, cooked, chowder (2)	359	(328–389)	37.0
King Crab, cooked, frozen	530		41.7
Lobster, cooked, frozen	376		31.6
Oyster, raw, frozen	154		30.8
Scallops, cooked, frozen (6)	335		31.9
Smelt, raw, frozen	237		28.2
Tuna, canned	334		34.4
Mean	348		36.6
Meat			
Beef and fat	492		14.7
Beef Liver	203		14.2
Chicken leg	255		12.6
Chicken wing	195		9.6
Ground round steak	258		8.9
Lamb chop (2)	249	(239–258)	13.3
Lamb liver	234		16.4
Pork loin	209		7.4
Top round steak	383		15.4
Gelatin, unflavored	283		35.8
Mean	267		14.8
Dairy products			
Cheese, cheddar (2)	268	(266–270)	6.3
Egg, whole	113		6.9
Egg, white	103		27.8
Egg, yolk (2)	101	(100–101)	2.9
Milk, whole, homog. vit. D	102		15.5
Milk, skim, powdered	1067		32.7
Milk, human (2)	29	(28–29)	3.8
Milk, fat free	162		46.3
Milk "cereal cream" 12% butter fat plus milk solids	280		6.9
Mean, less dry milk	157		18.2
Vegetables, fresh			
Leafy			
Broccoli	321		229.3
Brussels sprouts	233		145.6
Cabbage	166		63.8
Chicory	94		104.4
Chives	241		219.1
Endive	160		145.5
Escarole	89		80.9
Lettuce, green, outer	139		126.4
Lettuce, pale, inner	85		77.3
Parsley	566		269.5
Mean, less parsley	170		146.2

Table 22. (Continued)

	μg/g		mg/100 kcal
Vegetables, fresh			
Roots			
Carrots	185	(111–296)	80.4
Leek	264		105.6
Leek greens	478		191.2
Onion (2)	89	(75–103)	38.7
Parsnips	456		93.1
Potatoes (2)	167	(155–179)	19.2
Radish	162		103.0
Scallions	137		38.1
Turnip	95		52.8
Mean	226		80.8
Vegetables, fresh			
Legumes			
Beans, green, string	297		92.8
Beans, wax, yellow	241		89.3
Beans, Lima, dry (2)	1697	(1445–1948)	49.2
Beans, kidney, dry	775		22.4
Pencil pod, black, dry	1713		59.4
Peas, green	185		22.0
Pea, green, dry	1182		34.0
Mean, fresh	241		51.4
Vegetables, fresh			
Fleshy			
Artichoke, globe	487		324.7
Cauliflower	229		208.2
Celery	66		75.3
Cucumber	107		118.9
Egg plant	82		54.7
Mushroom	122		174.3
Pepper, green (3)	102	(92–114)	46.4
Squash, butternut	165		30.5
Squash, yellow	137		72.1
Squash, zuccini	237		139.4
Tomato (3)	177	(121–241)	126.4
Mean	174		124.4
Fruits and fruit juices			
Apple	58		12.9
Pear	64		16.0
Grape juice, canned	80		12.1
Orange juice	108		28.4
Papaya	0.1		<0.1
Mean, less papaya	78		17.4
Nuts			
Brazil nut	3175		49.3
Walnut	1315		24.0
Hazel nut	1739		29.0
Almond	2927		48.9
Peanut	1583		26.3
Pecan	1078		18.0
Mean	1970		32.6

Table 22. (Continued)

	µg/g		mg/100 kcal
Grains and cereal products			
Barley	1505		41.8
Buckwheat	2536		70.2
Corn, hybrid	664		69.2
Corn, Late sunshine	391		41.0
Corn, meal yellow	269		7.6
Corn starch	22		0.6
Cream filled doughnut	185		4.6
Kellogg's Special K	412		12.2
Kellogg's concentrate	574		15.6
Millet	1670		47.7
Egg noodles	702		19.9
Oats	1705		42.2
Puffed rice	150		4.3
Pumpernickel	550		22.0
Wheat, flour, patent	299		8.5
Wheat, gluten	18		0.5
Wheat bread	204		7.7
Whole wheat bread	340		13.8
Rice, brown	1477		40.9
Rice, wild	906		25.1
Rice, japanese (composite 204 samples)	251		7.0
Rye, whole	1333		39.8
Tapioca, minute	37		1.0
Wheaties	1032		23.9
Mean	805		23.6
Oil and fats			
Butter, lightly salted (2)	24	(20 27)	0.3
Butter, unsalted	6		< 0.1
Cod liver oil	1		–
Cottonseed oil	1		< 0.1
Peanut oil	8		0.1
Pork fat	5		< 0.1
Sunflower oil	6		< 0.1
Tallow, beef (2)	5	(2 8)	< 0.1
Mean	7		< 0.1
Condiments, spices, etc.			
Allspice, ground	1783		71.3
Cloves, whole	2549		101.9
Chili powder	3329		97.9
Garlic, fresh	230		16.8
Iodized salt	1667		–
Mustard, ground	4225		91.3
Pepper, black, ground	2323		75.2
Thyme, ground	615		24.6
Mean	2598		71.7
Beverages			
Beer	100		23.8
Cocoa, powdered	4289		94.9
Coffee, 97% caffein free, dry powder	6362	(5624-7143)	– –
Coffee, infusion	48		96.0
Gin	0.3		< 0.1
Grape wine, white	98		11.5

Table 22.(Continued)

	pCi/g		mg/100 kcal
Tea, black, infusion	11		110.0
Tea, black, leaves	2183		---
Tea, Japanese green, infusion	3		30.0
Tea, Japanese green, leaves	1666		---
Vermouth, Italian	135		9.9
Whisky, bourbon	1.3		< 0.1
Whisky, scotch	4.5		0.2
Sugars and syrups			
Corn syrup (3)	17	(14–23)	0.6
Honey	15		0.5
Jelly, mild cider	215		8.3
Jelly, wild cherry	32		1.2
Jelly, thyme-grape	33		1.3
Maple syrup, Vermont fancy (2)	150	(136–164)	5.1
Molasses, beet	110		4.3
Molasses, cane (4)	2093	(1311–2526)	81.4
Raw sugar, Columbian	122		3.1
Raw sugar, Phillipine	66		1.7
Sugar cane, green	190		---
Sugar crystals, cane, England	3		< 0.1
Sugar, white, crystals, U.S.	3		< 0.1
Sugar, white, France	2		< 0.1
Sugar, white, granulated, U.S.	2		< 0.1
Mean, less cane and molasses	59		2.0

Table 23. Annual Poundage Data for NAS Appendix A Substances (Groups I and II)

Substance name (Survey no.)	# Reports to NAS 1960/1970	Poundage reported to NAS (matching reports for both years) 1960	1970	Total 1970 poundage reported to NAS[a]
Magnesium carbonate NAS 0110	12/14	250,983	490,577	498,127
Magnesium hydroxide NAS 0111	---	---	---	170,116
Magnesium oxide NAS 0112	5/6	99	4,287	4,291
Magnesium phosphate (di) NAS 0113	---	4,400	12,855	12,855
Magnesium stearate NAS 0116	14/16	17,100	225,876	233,474
Magnesium sulfate NAS 0117	0/4	4,600	4,242	15,242

[a]There were no additional poundages reported to FEMA.

Other magnesium compounds and some of their uses include: magnesium chloride, as a color fixative, color retention agent, firming agent, and as an emulsifier for suspending dried skim milk in water (831); magnesium hydroxide, as an antacid, laxative, color fixative, color retention agent, and adjunct; magnesium oxide, as an antacid, laxative, buffer, neutralizing agent, and as a clarifier of sugar juice and reductant of molasses formation in raw sugar processing (610); magnesium phosphate (di- and tri-basic) and magnesium sulfate, as nutrients and dietary supplements. Magnesium sulfate has also been used in depression of free fatty acid formation in cold-stored fish muscle (677). Magnesium stearate is used as an anticaking and anti-drying agent, an emulsifier, a foaming and whipping agent, and as a binder, filler, or stabilizer for some plasticizers in the preparation of food-packaging materials. Magnesium stearate has also been used as a tablet lubricant, giving protection against the browning of spray-dried lactose in tablets (405). Table 24 shows usage levels, taken from an NAS/NRC survey, of some of these magnesium compounds. Table 25 also from an NAS/NRC survey, presents possible daily intake of these compounds.

Another source of exposure to magnesium is in water, where magnesium salts are partially responsible for the hardness of water. For a breakdown of magnesium concentrations in some American waters, consult Table 4 Chemical Section, VIII, Occurrence.

Table 24. Usage Levels Reported for NAS Appendix A Substances (Group I) Used in Regular Foods (R)

Substance name (Survey no.)	Food no.	Category name	# Firms reporting	- Usual use - WTD mean, %	- Maximum use - WTD mean, %
Magnesium carbonate NAS 0110	01	Baked goods (R)	-	.23393	.23394
	03	Other grain (R)	-	.00018	.00036
	07	Frozn dairy (R)	-	.01100	.01100
	10	Meat prods (R)	-	.00098	.00196
	11	Poultry (R)	-	.00100	.00200
	13	Fish prods (R)	-	.00200	.00200
	14	Procsd vegs (R)	-	---	---
	21	Soups (R)	-	.00100	.00200
	22	Snack foods (R)	-	.00100	.00200
	23	Bev type I (R)	-	.00641	.00641
	27	Gravies (R)	-	.02000	.02500
	30	Hard candy (R)	-	.00060	.00060
	48	Seas flavrs (R)	-	.90963	.96977
Magnesium hydroxide NAS 0111	05	Milk prods (R)	-	.07200	.09600
Magnesium oxide NAS 0112	06	Cheese (R)	-	.01000	.02000
	14	Procsd vegs (R)	-	.00380	---
	22	Snack foods (R)	-	.02000	.02000
	28	Imit dairy (R)	-	.00900	.00900
	30	Hard candy (R)	-	.08333	.08333
Magnesium phosphate di NAS 0113	16	Soft candy (R)	-	.03000	.03000
Magnesium stearate NAS 0116	01	Baked goods (R)	4	.00022	.00043
	16	Soft candy (R)	6	.66721	1.55070
	17	Conf frost (R)	-	.60404	.61129
	31	Chewing gum (R)	-	---	---
	48	Seas flavrs (R)	-	1.00000	1.00000

Table (Continued)

Substance name (Survey no.)	Food no.	Category name	# Firms reporting	- Usual use - WTD mean, %	- Maximum use - WTD mean, %
Magnesium sulfate	15	Condm relsh (R)	-	.00400	.00500
NAS 0117	23	Bev type I (R)	-	.01092	.01092
Malic acid	01	Baked goods (R)	7	.00997	.01193
NAS 0118 FEMA 2655	02	Break cerls (R)	4	.12082	.14355

Table 25. Possible Daily Intake of NAS Appendix A Substances (Groups I and II), per Food Category and Total Dietary, Based on Food Consumption by Total Sample -- See Explanatory Notes in Exhibits Section

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium carbonate NAS 0110	01	Baked goods (R)	-	0-5 mo.	7.953620	10.526850	7.953960
				6-11 mo.	59.418220	121.175740	59.420760
				12-23 mo.	127.491850	210.069140	127.497300
				2-65+ yr.	320.951960	476.749340	320.965680
Magnesium carbonate NAS 0110	03	Other grain (R)	-	0-5 mo.	.000900	.003000	.001800
				6-11 mo.	.017460	.051430	.034920
				12-23 mo.	.029520	.068220	.059040
				2-65+ yr.	.050040	.110520	.100080
Magnesium carbonate NAS 0110	07	Frozn dairy (R)	-	0-5 mo.	.110000	.451000	.110000
				6-11 mo.	1.045000	2.904000	1.045000
				12-23 mo.	1.584000	3.718000	1.584000
				2-65+ yr.	2.816000	6.787000	2.816000
Magnesium carbonate NAS 0110	10	Meat prods (R)	-	0-5 mo.	.010780	.028420	.021560
				6-11 mo.	.202860	.546840	.405720
				12-23 mo.	.295960	.508620	.591920
				2-65+ yr.	.768320	1.274980	1.536640
Magnesium carbonate NAS 0110	11	Poultry (R)	-	0-5 mo.	.005000	.023000	.010000
				6-11 mo.	.039000	.132000	.078000
				12-23 mo.	.066000	.184000	.132000
				2-65+ yr.	.129000	.328000	.258000
Magnesium carbonate NAS 0110	13	Fish prods (R)	-	0-5 mo.	.002000	.006000	.002000
				6-11 mo.	.026000	.098000	.026000
				12-23 mo.	.108000	.270000	.108000
				2-65+ yr.	.248000	.618000	.248000

Table 25. (Continued)

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium carbonate NAS 0110	14	Procsd vegs (R)	-	0-5 mo.	---	---	---
				6-11 mo.	---	---	---
				12-23 mo.	---	---	---
				2-65+ yr.	---	---	---
Magnesium carbonate NAS 0110	21	Soups (R)	-	0-5 mo.	.002000	.015000	.004000
				6-11 mo.	.233000	.727000	.466000
				12-23 mo.	.348000	.961000	.696000
				2-65+ yr.	.317000	.845000	.634000
Magnesium carbonate NAS 0110	22	Snack foods (R)	-	0-5 mo.	---	.001000	---
				6-11 mo.	.004000	.011000	.008000
				12-23 mo.	.011000	.031000	.022000
				2-65+ yr.	.013000	.037000	.026000
Magnesium carbonate NAS 0110	23	Bev type I (R)	-	0-5 mo.	.153840	.230760	.153040
				6-11 mo.	1.455070	4.980570	1.455070
				12-23 mo.	3.474220	10.416250	3.474220
				2-65+ yr.	6.666400	17.800570	6.666400
Magnesium carbonate NAS 0110	27	Gravies (R)	-	0-5 mo.	.020000	.060000	.025000
				6-11 mo.	.280000	.780000	.350000
				12-23 mo.	.720000	2.040000	.900000
				2-65+ yr.	1.660000	4.260000	2.075000
Magnesium carbonate NAS 0110	30	Hard candy (R)	-	0-5 mo.	.000000	.000000	.000000
				6-11 mo.	.000600	.001800	.000600
				12-23 mo.	.001800	.005400	.001800
				2-65+ yr.	.003600	.010200	.003600

Table 25. (Continued)

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium carbonate NAS 0110	48	Seas flavrs (R)	-	0-5 mo.	---	---	---
				6-11 mo.	---	.090963	---
				12-23 mo.	---	.181926	---
				2-65+ yr.	.090963	.454815	.096977
Magnesium carbonate NAS 0110		All categories	13	0-5 mo.	8.258140	11.345090	8.282160
				6-11 mo.	62.721210	131.499393	63.290070
				12-23 mo.	134.130350	228.453556	135.066280
				2-65+ yr.	333.714283	509.275425	335.426377
Magnesium hydroxide NAS 0111	05	Milk prods (R)	-	0-5 mo.	3.888000	2.880000	5.184000
				6-11 mo.	44.928000	216.072000	59.904000
				12-23 mo.	39.240000	125.568000	52.320000
				2-65+ yr.	28.440000	86.832000	37.920000
Magnesium hydroxide NAS 0111		All categories	-	0-5 mo.	3.888000	2.880000	5.184000
				6-11 mo.	44.928000	216.072000	59.904000
				12-23 mo.	39.240000	125.568000	52.320000
				2-65+ yr.	28.440000	86.832000	37.920000
Magnesium oxide NAS 0112	06	Cheese (R)	-	0-5 mo.	---	.010000	---
				6-11 mo.	.270000	.970000	.540000
				12-23 mo.	.780000	2.220000	1.560000
				2-65+ yr.	.940000	2.360000	1.880000
Magnesium oxide NAS 0112	14	Procsd vegs (R)	-	0-5 mo.	.053200	.159600	---
				6-11 mo.	.912000	2.128000	---
				12-23 mo.	1.482000	2.481400	---
				2-65+ yr.	3.230000	5.441600	---

Table 25 (Continued)

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium oxide NAS 0112	22	Snack foods (R)	-	0-5 mo.	---	.020000	---
				6-11 mo.	.080000	.220000	.080000
				12-23 mo.	.220000	.620000	.220000
				2-65+ yr.	.260000	.740000	.260000
Magnesium oxide NAS 0112	28	Imit dairy (R)	-	0-5 mo.	.000000	.000000	.000000
				6-11 mo.	.126000	.207000	.126000
				12-23 mo.	.072000	.306000	.072000
				2-65+ yr.	.081000	.135000	.081000
Magnesium oxide NAS 0112	30	Hard candy (R)	-	0-5 mo.	.000000	.000000	.000000
				6-11 mo.	.083330	.249990	.083330
				12-23 mo.	.249990	.749970	.249990
				2-65+ yr.	.499980	1.416610	.499980
Magnesium oxide NAS 0112	83	Formulas (B)	-	0-5 mo.	30.145860	55.227000	30.145860
				6-11 mo.	6.142320	29.265820	6.142320
				12-23 mo.	1.975600	.556760	1.975600
Magnexium oxide NAS 0112		All categories	7	0-5 mo.	30.199060	55.416600	30.145860
				6-11 mo.	7.613650	33.040810	6.971650
				12-23 mo.	4.779590	6.934130	4.077590
				2-65+ yr.	5.010980	10.093210	2.720980
Magnesium phosphate di NAS 0113	16	Soft candy (R)	-	0-5 mo.	.060000	.600000	.060000
				6-11 mo.	.660000	2.040000	.660000
				12-23 mo.	1.050000	2.790000	1.050000
				2-65+ yr.	1.740000	5.280000	1.740000

Table 25. (Continued)

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium phosphate di NAS 0113	83	Formulas (B)	-	0-5 mo.	1106.903610	2027.839500	1106.903610
				6-11 mo.	225.535320	1074.590070	225.535320
				12-23 mo.	72.540600	20.443260	72.540600
Magnesium phosphate di NAS 0113		All categories	-	0-5 mo.	1106.963610	2028.439500	1106.963610
				6-11 mo.	226.195320	1076.630070	226.195320
				12-23 mo.	73.590600	23.233260	73.590600
				2-65+ yr.	1.740000	5.280000	1.740000
Magnesium silicate NAS 0115	01	Baked goods (R)	-	0-5 mo.	.029920	.039600	.116620
				6-11 mo.	.223520	.455840	.871220
				12-23 mo.	.479600	.790240	1.869350
				2-65+ yr.	1.207360	1.793440	4.705960
Magnesium silicate NAS 0115	03	Other grain (R)	-	0-5 mo.	.012500	.042500	.015000
				6-11 mo.	.242500	.715000	.291000
				12-23 mo.	.410000	.947500	.492000
				2-65+ yr.	.695000	1.535000	.834000
Magnesium silicate NAS 0115	16	Soft candy (R)	-	0-5 mo.	.001400	.014000	.001400
				6-11 mo.	.015400	.047600	.015400
				12-23 mo.	.024500	.065100	.024500
				2-65+ yr.	.040600	.123200	.040600
Magnesium silicate NAS 0115	31	Chewing gum (R)	-	0-5 mo.	---	---	---
				6-11 mo.	---	---	---
				12-23 mo.	---	---	---
				2-65+ yr.	---	---	---

Table 2. (Continued)

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium silicate NAS 0115		All categories	5	0-5 mo.	.043820	.096100	.133020
				6-11 mo.	.481420	1.218440	1.177620
				12-23 mo.	.914100	1.802840	2.385850
				2-65+ yr.	1.942960	3.451640	5.580560
Magnesium stearate NAS 0116	01	Baked goods (R)	4	0-5 mo.	.007480	.009900	.014620
				6-11 mo.	.055880	.113960	.109220
				12-23 mo.	.119900	.197560	.234350
				2-65+ yr.	.301840	.448360	.589960
Magnesium stearate NAS 0116	16	Soft candy (R)	6	0-5 mo.	1.334420	13.344200	3.101400
				6-11 mo.	14.678620	45.370280	34.115400
				12-23 mo.	23.352350	62.050530	54.274500
				2-65+ yr.	38.698180	117.428960	89.940600
Magnesium stearate NAS 0116	17	Conf frost (R)	-	0-5 mo.	---	.604040	---
				6-11 mo.	.604040	1.208080	.611290
				12-23 mo.	1.208080	4.228280	1.222580
				2-65+ yr.	1.812120	4.832320	1.833870
Magnesium stearate NAS 0116	31	Chewing gum (R)	-	0-5 mo.	---	---	---
				6-11 mo.	---	---	---
				12-23 mo.	---	---	---
				2-65+ yr.	---	---	---
Magnesium stearate NAS 0116	48	Seas flavrs (R)	-	0-5 mo.	---	---	---
				6-11 mo.	---	.100000	---
				12-23 mo.	---	.200000	---
				2-65+ yr.	.100000	.500000	.100000

Table 25. (Continued)

Substances names (survey no.)	Food no.	Category name	# of Firms	Age	Possible daily intake, mg Average	High A	High B
Magnesium stearate NAS 0116		All categories	14	0-5 mo.	1.341900	13.958140	3.116020
				6-11 mo.	15.338540	46.792320	34.835910
				12-23 mo.	24.680330	66.676370	55.731430
				2-65+ yr.	40.912140	123.209640	92.464430
Magnesium sulfate NAS 0117	15	Condm relsh (R)	-	0-5 mo.	---	.004000	---
				6-11 mo.	.032000	.088000	.040000
				12-23 mo.	.112000	.304000	.140000
				2-65+ yr.	.352000	.848000	.440000
Magnesium sulfate NAS 0117	23	Bev type I (R)	-	0-5 mo.	.262080	.393120	.262080
				6-11 mo.	2.478840	8.484840	2.478840
				12-23 mo.	5.918640	17.745000	5.918640
				2-65+ yr.	11.356800	30.324840	11.356800
Magnesium sulfate NAS 0117		All categories	-	0-5 mo.	.262080	.397120	.262080
				6-11 mo.	2.510840	8.572840	2.518840
				12-23 mo.	6.030640	18.049000	6.058640
				2-65+ yr.	11.708800	31.172840	11.796800

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